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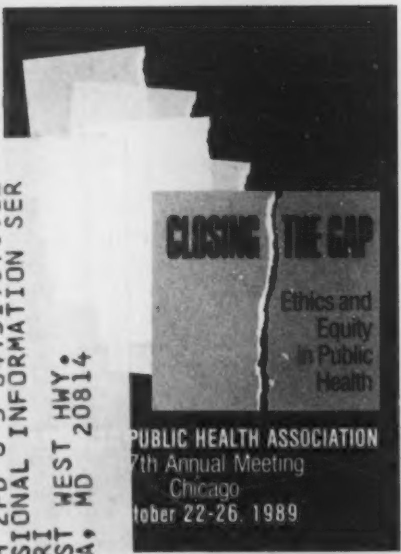

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Quality of Death Certificates in Valencia, Spain

Fernando G. Benavides, MD, PhD, Francisco Bolumar, MD, PhD, and Rafael Peris, MD, PhD

Abstract: Certificates of 1,454 deaths occurring over 11 months were retrieved from the Civil Register in Valencia, Spain. Relevant medical information was systematically gathered from hospital records, questionnaires, and Coroner reports. We compared the underlying cause of death (UCD) from the original death certificate, and a reference cause of death (RCD) determined by a panel of experts based on all available information. Overall, 80.2 percent of the certificates were concordant for disease category, but there was a great disparity among some specific disease categories. (*Am J Public Health* 1989; 79:1352–1354.)

Introduction

The quality of mortality statistics has been studied frequently.[1–6]

While mortality statistics are commonly used both for research and administrative purposes in Spain, their reliability has not yet been studied in full detail. This is an important omission; the first studies in this field were carried out in other countries as early as the 1940s.[7–9]

To date, studies of the quality of the registered causes of death have been based on death certificates selected either because stated causes were specific diseases or because of the availability of information required to test the concordance of the death certificate.[10–14] In order to minimize such selection biases, the present investigation was population-based.

Methods

All death certificates for the 255,866 residents in the catchment area of the Teaching Hospital in the region of Valencia, Spain from February–December 1984 were obtained from the Civil Register. For the resulting 1,454 certifications, we systematically reviewed autopsy findings, questionnaires addressed to the certifiers, and hospital records.

The monthly search for this information started in the Teaching Hospital. If hospital records corresponding to each death were available in the first week of the search, and the last inpatient summary had been completed within the three months prior to death, the search for clinical information was concluded.

When results of first step were negative, a questionnaire (based on work by Moriyama, *et al.*[15]) requesting clinical information about the death was sent to the certifier in the first week of the month following the death, and a list of all such deaths was sent to the Hospital Records departments of all the other hospitals in the city.

Finally, records were obtained from the Coroner's Office for those deaths which had been certified by a Coroner.

All the available information was reviewed separately by two specialists in Internal Medicine, who were not conversant with the information recorded in the original death certificate. Kappa index of agreement between the two reviewers was 0.78 (confidence intervals 0.76, 0.81). When internists opinions did not coincide, a third specialist was called in to review the information and thus provide a conclusive decision. All three specialists had been trained previously in certifying procedures by experienced physicians from the Office of Mortality Statistics.

Applying the rules of the ICD-9th revision,[16] professional coders selected the underlying cause of death (UCD) from each original certificate and the reference cause of death (RCD) from each standard certificate produced by the internists.

Using two by two tables for each cause and for each group of causes, the Detection Rate (DR),* and the Confirmation Rate (CR),** were computed working at a three-digit level of ICD-9th codes.

From the initial 1,454 certificates, clinical information was obtained for 1,176 deaths; in the remaining 278 deaths neither clinical records were available nor were questionnaires answered. Out of the 1,176 deaths, 108 (9.2 percent) were not included in the study because the medical reviewers considered the clinical information inadequate to test the concordance. Therefore, the final number of deaths studied was 1,068 (73.5 percent of the initial sample).

Results

In 50.9 percent of the 1,068 deaths, clinical information was obtained only from hospital records, in 27.7 percent only from questionnaires, and in 11.9 percent from both sources. Coroners' reports provided the remaining 9.4 percent of the information, of which 1.9 percent had also hospital records. The total number of deaths with hospital records amounted to 692. In 40 cases, autopsies were carried out by hospital pathologists. Of the 692 deaths with information obtained through hospital records, 562 (81.2 percent) took place in hospitals.

According to the completed questionnaires, the duration of the professional relationship between the certifier and the deceased had lasted less than 24 hours in 29.2 percent of the cases. When deaths occurred in hospitals, this percentage increased to 53.7 percent; when deaths occurred at home, it decreased to 17.9 percent.

Agreement between Detection and Confirmation Rates did not vary by duration of professional relationship, by place

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Editor's Note: See also related editorial p 1349 this issue.

*Detection Rate equals the number of concordant deaths according to a particular disease category divided by the number of standard death certificates from the same disease category.

**Confirmation Rate equals the number of concordant deaths according to a particular disease category divided by the number of original death certificates from the same disease category.

TABLE 1—Percentage Agreement (with 95% Confidence Intervals) of Detection and Confirmation Rates by Place of Death, Duration of Professional Relationship and Sources of Information

Place of death	N	Agreement Indexes (%)	95% CI
Home	426	80.5	76.7, 84.2
Hospital	642	80.1	77.0, 83.1
Duration of professional relationship			
>3 months	215	81.8	76.7, 86.9
<3 months	85	82.4	74.3, 95.5
<24 hours	124	81.5	74.6, 88.3
Sources of information			
Clinical records	692	79.1	76.1, 82.1
Questionnaires (only)	296	82.1	77.7, 86.5

of death, and by sources of information (Table 1).

The agreement indexes for all causes were 80.2 percent both for the detection and confirmation rates, as shown in Table 2.

The agreement indexes, DR and CR, for the major categories of causes of death can also be found in Table 2. They are highest for tumors (DR = 89.9 percent and CR = 95.3 percent) and circulatory diseases (DR = 87.6 percent and CR = 80.5 percent).

Agreement indexes for the rest of groups of causes were questionable, due to the small number of deaths on which they were based. Table 3 displays the matrix plotting underlying cause against reference cause.

As regards specific tumor sites, the highest indexes of agreement were those for lung cancer (DR = 84.6 percent and CR = 91.7 percent) and breast cancer (DR = 88 percent and CR = 95.7 percent). Conversely, liver cancer showed very low rates of agreement (DR = 53.8 percent and CR = 37.9 percent), and colon cancer showed indexes below 80 percent but of a very similar nature (DR = 75 percent and CR = 71.4 percent).*

Agreement indexes for circulatory diseases varied greatly. Hypertensive disease produced low indexes of agreement. Arteriosclerosis gave quite similar results, with even lower indexes (DR = 19.2 percent and CR = 21.7 percent). Acute myocardial infarction showed a DR of 75.5 percent and a CR of 53.7 percent and the intra craneal haemorrhage a DR of 84.2 percent and a CR of 55.2 percent. However, the overall agreement in this group was over 80 percent.*

Discussion

In these data, mortality statistics as a whole appear to be reasonably reliable: 80.2 percent of the certificates showed good concordance. This figure lies somewhere within the range of results obtained by Alderson[4] in the Oxford Record Linkage Study Area (61 percent of agreement) and by Faire[5] among Swedish twins born in 1901–25 (DR = 94.9 percent and CR = 95.6 percent) (our estimate from their figures).

When comparing our population based results with those of Kircher in Connecticut,[14] based on autopsies, we found his confirmation rates to be lower than ours for neoplasms, circulatory diseases, and respiratory diseases (85 percent versus 95.3 percent; 75 percent versus 85.5 percent and 31 percent versus 60.3 percent, respectively). These differences might be explained, at least to some extent, by the fact that when only autopsies are used to validate death certificates, a higher level of disagreement is to be expected as autopsies are carried out mainly on diagnoses that appear uncertain.

It is interesting to note that in our study 13 deaths initially certified as due to cancer were reclassified after reviewing the available clinical information, and 30 deaths initially assigned to groups other than tumors were then reclassified as due to cancer. This finding suggests that mortality from cancer was underreported by 6.1 percent, a figure that lies between the value of 5 percent found by Griffith in England and Wales[17] and the value of 10 percent observed by Engel in Georgia.[18]

Percy, *et al,*[6] estimate a DR and CR of 82.7 percent for tumors in the USA, a value close to ours as regards DR but lower in respect to CR. Our results are relatively close to those obtained by Faire[5] in Sweden with a DR of 98.2 percent and a CR of 99.5 percent.

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*Data available on request to author.

TABLE 2—Agreement Indexes for the Underlying Causes of Death, ICD-9A Main Category

Disease Category	UCD(*) N	RCD(**) N	Agreements N	Detection Rate %	Detection Rate SD	Confirmation Rate %	Confirmation Rate SD
Infectious/parasitic	6	7	2	28.6	17.1	33.3	19.2
Neoplasm	279	296	266	89.9	1.8	95.3	1.3
Endocrine/metabolic	43	51	30	58.8	6.9	68.8	7.0
Blood organs	4	1	1	100.0	0.0	25.0	21.7
Mental disorders	7	3	2	66.7	27.2	28.6	17.1
Nervous system	13	8	6	75.0	15.3	46.2	13.8
Circulatory	430	395	346	87.6	1.7	80.5	1.9
Respiratory	78	85	47	55.3	5.4	60.3	5.5
Digestive	83	99	73	73.7	4.4	87.5	3.6
Genitourinary	29	28	14	50.0	9.5	48.3	9.3
Pregnancy	1	1	1	100.0	0.0	100.0	0.0
Skin	0	2	0	0.0	0.0	0.0	0.0
Musculoskeletal	1	2	1	50.0	35.4	100.0	0.0
Congenital anomalies	7	6	6	100.0	0.0	85.7	13.2
Perinatal	3	4	3	75.0	21.7	100.0	0.0
Ill-defined	14	1	0	0.0	0.0	0.0	0.0
External causes	70	79	59	74.7	4.9	84.3	4.4
Total	1068	1068	857	80.2	1.2	80.2	1.2

(*)UCD: Underlying Cause of Death
(**)RCD: Reference Cause of death.

TABLE 3—Agreement Matrix between Underlying Cause of Death and Reference Cause of Death (RCD)

Underlying Cause of Death	ICD-9*	Reference Cause of Death 001 −139	140 −239	240 −279	280 −289	290 −319	320 −389	390 −459	460 −519	520 −579	580 −629	630 −676	680 −709	710 −739	740 −759	760 −779	780 −799	E800 −E999	Total U.C.D.
Infectious/parasitic	(001–139)	2	2							2									6
Neoplasm	(140–239)	1	266	1				3		5	2			1					279
Endocrine-metabolic	(240–279)		2	30				8		3									43
Blood organs	(280–289)		1		1			1		1									4
Mental disorders	(290–319)					2		4			1								7
Nervous system	(320–389)			1			6	4			1							1	13
Circulatory	(390–459)	1	14	15		1	1	346	26	9	7					1		9	430
Respiratory	(460–519)	1	4				1	12	47	4	1		1				1	6	78
Digestive	(520–579)		4	2					3	73			1						83
Genitourinary	(580–629)		3	2				8			14							2	29
Pregnancy	(630–676)											1							1
Skin	(680–709)																		0
Musculoskeletal	(710–739)													1					1
Congenital anomalies	(740–759)							1							6				7
Perinatal	(760–779)															3			3
Ill-defined	(780–799)							4	7		1							2	14
External causes	(E800–E999)	2						4	2	2	1							59	70
Total RCD		7	296	51	1	3	8	395	85	99	28	1	2	2	6	4	1	79	1068

With respect to mortality due to cardiovascular diseases, our indexes lie between those obtained by Engel[18] (DR = 69.0; CR = 75.6) and those found by Faire[5] in Sweden (DR = 92.4; CR = 88.8).

It has been argued that concordance indexes might depend on the precision and completeness of the information used by the reviewers to establish the standard cause of death. In our case, the influence of what has been called reviewer bias does not seem to play an important role, as additional information obtained after the death usually comes from autopsies and the number of these in our area was dramatically low.

It has also been argued that reviewers might have access to clinical records to which medical certifiers did not have access. But this does not accord with the fact that in our study 81.2 percent of the deaths with clinical records available took place and were certified in hospitals. However, even if this were the case, it would lead to a real discrepancy since the reviewers would have been able to establish the cause of death more accurately.

On the contrary, if the medical certifiers had access, the discrepancies would be spurious. Nevertheless, 72.3 percent of the information used to test the degree of concordance came from hospital records and forensic reports and thus was equally available for both certifiers and reviewers.

Some information was obtained only from questionnaires. This presented the problem of the reviewing doctors not having access to any more information than that which was transcribed. However for 70.5 percent of the completed questionnaires, corresponding to deaths at home, there was presumably no more information than that which appeared on the questionnaires, as the systematic search for the clinical records of these cases was negative. However, the possibility that a crucial piece of information which might have been known to the medical certifier did not appear in the records, although remote, cannot be ruled out in this sort of study.

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Risk Reduction among Intravenous Drug Users in Amsterdam under the Influence of AIDS

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Abstract: We evaluated the impact of a needle/syringe exchange program among 263 drug users in the period December 1985 to April 1988. Participants in this study were asked about their risk-behavior at three different visits. We found no increase in the proportion injecting drugs or in the frequency of intravenous drug use. A strong decrease in borrowing and lending of used needles/syringes was found and this behavioral change was not dependent on learning human immunodeficiency virus (HIV) serostatus. Although use of the exchange program increased over time, reduction in needle sharing was mainly an effect of the study with only a limited impact of the exchange program. We conclude that ample provision of needles and syringes is an important starting point but in itself not enough to produce the necessary drastic change in risk behavior. Intensive counseling of the drug user is also needed. (*Am J Public Health* 1989; 79:1355–1357.)

Introduction

In the absence of an effective treatment or vaccine, control of infections with the human immunodeficiency virus (HIV) relies on efforts to reduce risk behavior. To control the spread of HIV among intravenous drug users (IVDUs) a prevention campaign was launched in Amsterdam, The Netherlands.[1]

Leaflets with information on HIV and its transmission are distributed and meetings are held for workers in the drug field and for drug users. As sharing of needles and syringes is considered to be the main transmission route among intravenous drug users,[2] a needle/syringe exchange program was organized as part of this campaign. The exchange program started on a small scale in 1984; by 1987 700,000 syringes and needles were provided through existing social agencies and treatment facilities.[3] To evaluate the impact of the campaign, a longitudinal study was undertaken. The objectives of this study are to determine whether drug users are able to change their risk behavior and if risk reduction occurred, whether this can be ascribed to the prevention campaign or to the effect of serological testing and counseling during the study.

Methods

Participants were recruited at six methadone outposts and the weekly sexually transmitted disease clinic for drug-using prostitutes. None of these facilities are drug free. As one of the aims of the study was to find out whether the syringe exchange program could lead to an increase in intravenous drug use, ex-intravenous drug users and nonintravenous drug users were also invited to participate.

Participation was voluntary and, after extensive information had been given, informed consent was obtained. A blood sample was taken and participants interviewed using a standard questionnaire which included questions concerning clinical symptoms, medical history, lifestyle, use of oral and intravenous drugs (methadone included), and prostitution. Participants were asked to return for a follow-up visit every four months. At each occasion, the same interview and a blood sample were taken. To encourage continued participation 25 Dutch guilders ($12 US) were paid per follow-up visit.

From December 1985 to March 1988, 569 drug users without HIV-related disease entered the study, approximately one-seventh of the drug users visiting the methadone posts and the special STD-clinic in that period; 263 (46 percent) returned for at least one follow-up visit before July 1988. No difference was found in age, gender, nationality, and history of prostitution between the 263 drug users who returned and the 306 who did not return for a follow-up visit. However, among those who returned, a higher percentage was infected with HIV (33.1 percent vs 19.0 percent, 95% CI of difference = 6.9, 21.3), had been staying in Amsterdam longer than one year (88.2 percent vs 78.4 percent, 95% CI of difference = 3.8, 15.8), and had a history of current IV drug use (71.9 percent vs 53.9 percent, 95% CI of difference = 10.2, 25.8).

We compared results of the interview regarding the intravenous drug use behavior in the six-month period preceding the intake and during the follow-up period.

Since participants did not return for follow-up regularly, only those with an interval of three to six months between the visits were selected for study. As a result, 233 participants remained whose second visit data could be analyzed. The mean period between the first and second visit was 127 days (SD ± 20). A sub-group of 165 participants made a third visit, enabling us to study the risk behavior over a longer period of time. The mean duration between the second and third visit was 123 days (SD ± 20). There was no difference in age, gender, duration of drug use, nationality, and HIV-prevalence between the first-, second- and third-visit groups.

To determine the effect of infection status on risk behavior, three different groups were distinguished among 140 current intravenous drug users: those participants who did not know their serostatus (17); those who knew that they were infected with HIV (39); and those who knew that they were uninfected (84). Forty-nine other current users were excluded because knowledge of serostatus was too close to their second visit or no information was available about the date of disclosure.

The average time between intake and disclosure of the serostatus was 32 days (SD ± 7), and 97 days (SD ± 19) between disclosure and second visit. Only data of the first and second visit were studied; at the third visit the number of persons available was too small.

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Recruitment into the study took place over a period of 27 months. Thus we could also study the risk behavior of current intravenous drug users at intake over time. We divided the 27-month intake period into four periods of equal length (207 days). The HIV antibody seroprevalence of the four intake-groups defined their risk category.

χ^2 test with Yates' correction and χ^2 for trend in proportions were used for testing group differences and behavioral changes. Log-linear modeling[4] was employed for testing differences in behavioral changes between subgroups and for differentiating between effects on behavior caused by the prevention campaign (time effect) or by the study itself (study effect). Serological tests used for HIV-ab testing were ELISA (enzyme-linked immunosorbent assay) and confirmation of positive specimens was performed by immunoblotting and competitive ELISA.[5]

Results

Of the 263 study participants, 140 were male and 123 female. The mean age was 29.1 years (SD 5.4). Seventy-one percent were Dutch, 20 percent German, and 9 percent another nationality. Two hundred and thirty-three of the 263 participants had a history of intravenous drug use at entry into the study; 189 of them used drugs intravenously at least once during the six months preceding intake (current intravenous drug users). Heroin in combination with cocaine was injected by 58 percent of the current users, 21 percent used heroin alone, 7 percent cocaine alone, and 5 percent amphetamine alone. At entry into the study, 87 (33 percent) of the 263 participants had antibodies to HIV (36 percent of the men and 30 percent of the women). None of those 87 HIV-infected had severe HIV-related disease (acquired immunodeficiency syndrome or AIDS-related complex).

The proportion of current intravenous drug users who obtained their needles and syringes exclusively through the exchange program increased from 31.2 percent at the first visit to 52.3 percent at the third visit (Table 1), although the proportion of all drug users reporting using drugs intravenously and the frequency of injecting did not differ over time. Borrowing and lending needles and syringes among current users decreased sharply over time. The behavioral change occurred in both men and women, and in all nationality groups.* Decrease in needle sharing occurred to the same extent regardless of knowledge of serostatus (Figure 1).

The borrowing behavior at intake of current intravenous drug users was comparable over time although those who entered the study later lent their needles/syringes less often to others than those who entered in the beginning (Table 2). The use of the exchange system and single syringe use increased over time. The HIV-prevalence of those entering the study decreased over time.

To investigate the relative importance of a study-related (follow-up) effect versus a time-related (prevention campaign) effect, the effects were log linearly modeled and tested.* The time variable was dichotomized at the midpoint of the intake period.

The reduction in borrowing was merely a study effect, being unrelated to time. The likelihood ratio statistics of lending were significant for both study and time effects indicating a strong study-related reduction in lending behavior independent from the mere time effect. The increase in the use of the exchange system appeared mainly a time-related effect. The increase of single needle/syringe use appeared to be both study-related and time-related. These data are also suggested by inspection of Tables 1 and 2.

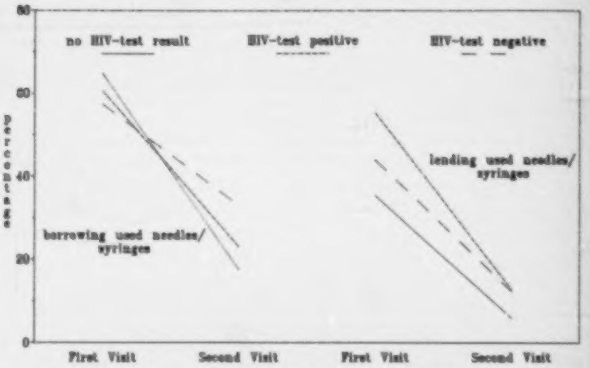

FIGURE 1—Current Intravenous Drug Users Engaged in Needle/Syringe Sharing by HIV-ab Test Result in Amsterdam, December 1985–July 1988

Discussion

The results of this follow-up study clearly show that IV drug users are able to reduce their sharing of needles/syringes, the main risk factor for contracting HIV in this group. Risk reduction among IV drug users has also been observed in New York City,[6] and in various retrospective studies.[7] A drawback of retrospective studies is that participants may tend to make the past look worse and the present better.

There was a sharp increase in the proportion of intravenous drug users in Amsterdam making exclusive use of the exchange program to obtain clean needles and syringes, apparently reflecting the effect of the prevention campaign.

We found no evidence that the non-intravenous drug users in our study group started intravenous use in spite of the availability of clean needles and syringes. Apparently the way in which drugs are used is rather stable.

Although an increasing number of intravenous drug users in Amsterdam obtain their needles/syringes through the exchange program, the impact of this prevention campaign on

TABLE 1—Current Intravenous Drug Users* Engaged in Specific Risk Behavior during Follow-up, Amsterdam, December 1985–July 1988

% of Current IVDU	First Visit n = 189	2nd Visit n = 156	3rd Visit n = 107	p-value**
Shooting daily	55	48	54	0.72
Shooting at home	79	80	80	0.75
Shooting alone	28	31	26	0.94
100% use of needle/syringe exchange program	31	40	52	0.0004
Single needle/syringe use	43	54	65	0.0003
Borrowing used needles/syringes ≥1×	56	26	16	10^{-6}
Lending used needles/syringes ≥1×	44	10	8	10^{-6}

*At the first visit 189/263 (72%) drug users were current intravenous drug users, second visit 156/233 (67%), third visit 107/165 (65%).

**χ^2 test for trend.

Data available on request to author.

TABLE 2—Risk Behavior of 189 Current Intravenous Drug Users at Intake (December 1985–March 1988) Divided by Intake Period

	Intake Period*				
IVDU Behavior	I (n = 54)	II (n = 46)	III (n = 56)	IV (n = 33)	p-value**
	%	%	%	%	
Exclusive use of exchange system	13.0	10.9	44.6	66.7	10^{-6}
Borrowing	55.6	58.7	56.4	46.9	0.51
Lending	50.9	53.3	34.5	34.4	0.04
Single syringe use	38.9	34.8	42.9	60.6	0.06
HIV-ab seropositive	50.0	37.0	37.5	24.2	0.02

*Each intake-period covers 207 days: I. 12/04/85–06/28/86
II. 06/29/86–01/21/87
III. 01/22/87–08/16/87
IV. 08/17/87–03/11/88

**χ^2 test for trend.

risk behavior still leaves something to be desired. Borrowing used needles and syringes—at least once in the prior six months—was as often reported by those who entered our study at the end in 1986 as it was in 1988, even though this last intake group had a lower HIV-prevalence indicating they had been less at risk for HIV-infection. As we asked only whether the participants borrowed at least once, it may be that the frequency of borrowing diminished over time.

However, to be effective, risk reduction has to be total. Such a strong behavioral change was found only among the intravenous drug users who participated in the follow-up study.

The extent of behavioral change that we found during follow-up did not depend on learning one's infection status. In contrast, studies of homosexual men have found that seropositives changed their behavior more than seronegatives and untested comparison groups.[8] Apparently, among drug users counseling has a more important influence. From our results it seems that the needle/syringe exchange program and a testing program on a voluntary basis can be expected to have strong influences on risk behavior only when such programs are combined with intensive counseling.

We conclude that the Amsterdam AIDS prevention campaign did not increase the intravenous use of drugs, as is often feared, but did not automatically lead to complete change of risk behavior among intravenous drug users. To obtain such a maximum effect, intensive counseling of drug users seems indicated. Proposals have now been formulated to start with two special teams of counselors, one for drug users who participate in the methadone programs and one for those drug users who are not in contact with treatment programs.

We recognize that we have studied risk reduction among a selected group that may not represent all drug users in Amsterdam, but this is inevitable in all studies of drug users. In addition, the results are based on self-reporting. An objective measure of self-reported risk reduction is a reduced attack rate of HIV infection over time. As of July 1988, we have observed 10 seroconversions among 148 seronegative drug users with a history of IV drug use in the previous five years. The attack-rate (Kaplan-Meier) after a follow-up of one year was 7.3 percent (95% CI: 2.6, 12.0). The number of seroconversions in the follow-up period is still too small to enable us to do a reliable comparative study of the attack rate over time in relation to this process of risk reduction.

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Impact of the AIDS Epidemic on Morbidity and Mortality among Intravenous Drug Users in a New York City Methadone Maintenance Program

Peter A. Selwyn, MD, MPH, Diana Hartel, MPH, William Wasserman, MPH, and Ernest Drucker, PhD

Abstract: To examine the impact of the AIDS epidemic on morbidity and mortality in a defined population of intravenous drug users, we analyzed overall and cause-specific death rates, AIDS incidence, and acute medical hospitalizations among patients in a long-term methadone maintenance program in New York City for the years 1984 through 1987 (midyear population for each year 828 to 891; demographic characteristics did not differ). The number of deaths while in treatment increased from 11 (13.3/1000) in 1984 to 39 (44.2/1000) in 1987. Deaths from AIDS increased from 3.6/1000 to 14.7/1000, deaths due to bacterial pneumonia/sepsis from 3.6/1000 to 13.6/1000; deaths from cirrhosis, drug overdose, trauma, and other causes remained relatively stable. AIDS incidence rose from six cases/1000 in 1984 to 20.4.1000 in 1987. Hospitalizations for AIDS, pneumonia, tuberculosis, and endocarditis/sepsis increased from 84.9/1000 in 1986 to 144.8/1000 in 1987.

These data suggest that the AIDS epidemic has had a profound effect on patterns of morbidity and mortality among intravenous drug users in this methadone program population. Drug treatment programs may be important sites for targeting clinical services for drug users with AIDS, although the increasing burden of AIDS-related disease will require expansion of existing funding and treatment resources. (*Am J Public Health* 1989; 79:1358–1362.)

Introduction

Intravenous drug users have historically been known to be at increased risk of death from drug overdose, alcoholism, trauma, and infectious causes.[1–4] Since the early 1980s, the acquired immunodeficiency syndrome (AIDS) has become a new and increasingly important cause of death among drug users, especially in New York City.[5–7] In addition, New York City surveillance data demonstrate a concurrent increase in pneumonia hospitalizations and deaths among narcotics addicts,[5,8,9] which may reflect additional consequences of human immunodeficiency virus (HIV) infection in this population.[5,7–11]

It has been estimated that there are between 1.1 and 1.3 million intravenous drug users in the United States, with over 200,000 in the New York City metropolitan area alone.[6,12] In New York State, there are currently over 35,000 patients enrolled in methadone maintenance treatment programs.* Historical patterns of morbidity and mortality among patients in methadone maintenance treatment are well described[13–19]; however, most of this literature predates the AIDS era and does not address the impact of the current epidemic of HIV infection and AIDS on such populations.

We examined AIDS incidence rates, overall and cause-specific mortality rates, and medical hospitalization patterns among a defined population of intravenous drug users in a long-term methadone maintenance treatment program in the Bronx, NY, from 1984 through 1987.

Methods

This study was conducted at the Drug Abuse Treatment Program of Montefiore Medical Center, Bronx, NY, which operates three outpatient methadone maintenance clinics for chronic opiate addicts. The treatment program, which serves a population of approximately 900 current and former intravenous drug users, provides on-site primary medical care for all patients, as well as methadone maintenance and support services.

In late 1983, centralized registries were established and maintained by program medical staff for recording all AIDS diagnoses and deaths among program patients; in late 1985, a log was established and reviewed weekly to monitor all hospitalizations occurring in the patient population. AIDS cases were identified both through ongoing patient contact with on-site medical services at the methadone program and through review of hospital records for suspected cases. Causes of death were identified and verified (by the first author) for all patients dying while in treatment, through a review of hospital records, death certificates, and medical examiners' reports for cases in which postmortem examinations were performed. Surveillance for hospitalizations was facilitated by inpatient follow-up of patients referred by medical staff for hospital admission, and by the requirement that hospital staff must contact clinic staff in order to verify methadone dosages for hospitalized patients.

Overall and cause-specific mortality rates per 1000 for methadone program patients dying while in treatment were calculated for each calendar year from 1984 through 1987, using the midyear population census as the denominator for each year. Person-time death rates were also calculated based on time spent enrolled in the methadone program and are expressed as deaths per 1000 person-years. Deaths were classified as due to AIDS in cases with either an ante- or postmortem AIDS diagnosis, in which AIDS was listed as a cause of death; deaths were ascribed to other causes only after AIDS diagnoses had been excluded through review of all available information. In order to evaluate the possible relation between HIV infection and causes of death other than AIDS itself (e.g., deaths from bacterial pneumonia or sepsis), we examined the HIV serologic status of the subgroup of deceased patients who had also been enrolled in a

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*New York State Division of Substance Abuse Services: Census, Capacity, Utilization and Waiting List Report, November 1988.

seroprevalence and longitudinal study of HIV infection initiated in 1985 at the same clinic site.[20] This ongoing study, aspects of which have been previously described,[20–22] involved voluntary, confidential HIV antibody testing and clinical/research follow-up for a cumulative cohort of over 600 patients in the Montefiore program. The overall HIV seroprevalence level in the cohort has been approximately 40 percent.[20–22] As described elsewhere, seroprevalence study participants and non-participants did not differ with respect to standard demographic variables or the length of time in methadone treatment.[20–22] While the aim of the seroprevalence and longitudinal study has been to examine the natural history of HIV infection, that of the current analysis was to evaluate the impact of the epidemic of AIDS and HIV infection over time on a defined population in drug abuse treatment.

The incidence of AIDS in the entire study population was calculated for each year in the four-year period, with the occurrence of incident cases of AIDS expressed both as a proportion per 1000 persons and as a rate per 1000 person-years in treatment. In addition to incident AIDS cases arising out of the clinic population, yearly trends in the number of patients already diagnosed with AIDS and referred to the program for drug abuse treatment were monitored. To ensure uniformity of definitions and reporting, the original[23] and not the revised[24] Centers for Disease Control (CDC) AIDS surveillance definition was used for all four years of the study period as the standard for AIDS diagnoses.

Medical hospitalization logs, which were available only for the latter two full years of the study period, were reviewed for the years 1986 and 1987. Hospitalizations were classified by primary diagnosis into the most frequently occurring categories, and the frequencies of hospitalization for different diagnostic categories were compared for the two 12-month periods. Diagnoses were confirmed through review of hospital records. Hospitalizations for all AIDS-specific opportunistic infections were classified as due to AIDS. Elective admissions for inpatient alcohol or drug detoxification were excluded from this analysis. Hospitalization events by year were calculated for each year with the total number of patients enrolled in the program each year as the denominator.

As noted above for the determination of AIDS incidence and death rates, only those events are included which occurred while patients were enrolled in the methadone program; the analysis does not reflect any additional outcomes which may have occurred among patients leaving the program during the study period. Estimates should therefore be considered minimum estimates. The midyear patient population was chosen as the denominator for incidence calculations for each year since there was movement in and out of the program but the number of persons entering and leaving treatment was balanced for each year of the study period. An examination of time spent by patients in the program each year indicated that person-time did not differ between years, with individuals remaining in the program for a mean of approximately 300 days each year. Analyses of changes in incidence rates over the four-year study period were based on point and interval estimates of rate differences.[25] In addition, trends over the four years in AIDS incidence and overall and cause-specific mortality rates were examined.

Results

From January 1, 1984 through December 31, 1987, a total of 1,598 patients were treated: 59 percent male, 20 percent White, 27 percent Black, 52 percent Hispanic (primarily Puerto Rican), and 1 percent classified as "other." Racial and gender distributions did not differ by year for any of the four years. The median age of the entire patient population as of January 1, 1984, was 32 years, with the central three-fourths of all patients each year between the ages of 30 and 45. The overall age distribution (by year of birth) was fairly constant from year to year. The midyear patient census for each of the four years ranged from 828 to 891 (Table 1). Of the 1,598 patients treated in the program during the study period, 844 (53 percent) were enrolled prior to 1984, of whom 416 (49 percent) remained continuously enrolled through the end of 1987. Seven hundred and six patients were newly admitted during the four years, and 48 patients were readmitted after having left treatment before 1984. The number of patients discharged from treatment each year ranged from 162 to 197; discharged patients did not differ by year with respect to race, gender, or age, nor was there a statistically significant trend in drop-out rates during the study period.

Death Rates

A total of 94 patients died while in treatment during the study period (Table 1). The yearly number of deaths increased steadily from 1984 through 1987, even though the midyear population each year remained stable. The proportion dying (based on the mid-year population size) increased from 13.3/1000 in 1984 to 44.2/1000 in 1987. By person-time analysis, there were 12.9 deaths/1000 person-years in 1984, compared with 44.0/1000 person-years in 1987.

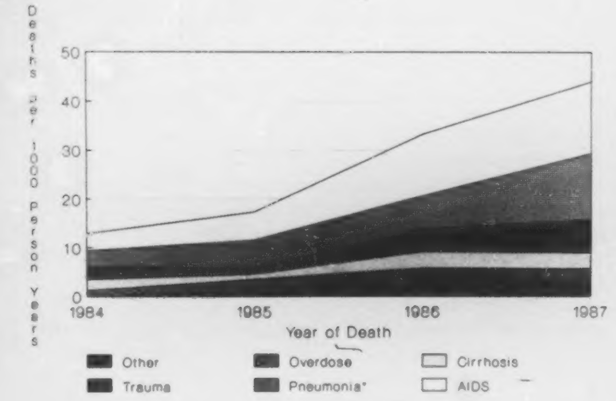
Causes of Death

The crude mortality rate from AIDS in 1984 was 3.6 per 1000 patients. This rate increased steadily through the study period to 14.7 per 1000 in 1987. Deaths from pneumonia, endocarditis, and bacterial sepsis combined—in patients without any diagnosis of AIDS—also rose from 3.6 per 1000 in 1984 to 13.6 per 1000 in 1987. Smaller or minimal increases were seen for deaths from cirrhosis, drug overdose, trauma, and other causes, which were less frequent causes of death. Cause-specific death rates (person-time) among methadone program patients are graphically displayed in Figure 1. The number of deaths due to AIDS rose from 3.5/1000 person-years in 1984 to 14.7/1000 person-years in 1987 (difference 11.2/1000, 95% CI: 2.2, 20.0). Similarly, there was an increase in death rates from pneumonia, endocarditis, and bacterial sepsis, which together rose from 3.5 to 13.5/1000 person-years from the first to the fourth year of the study period (difference = 10.0, 95% CI: 1.4, 18.6). Deaths due to alcoholism/cirrhosis and drug overdose remained relatively constant between 1984 and 1987 (3.5 to 5.7/1000 person-years); death rates from trauma, including homicide, suicide, and vehicular trauma, showed a slight increase during the

TABLE 1—Deaths by Year among Montefiore Methadone Program Patients, New York City, 1984–87

	1984	1985	1986	1987
Midyear Census	828	875	891	882
Person-years	850	864	873	886
Mean Days Observed	296	300	294	300
Number of Deaths	11	15	29	39
Deaths per 1000	13.3	17.1	32.5	44.2
Deaths/1000 person-yrs	12.9	17.4	33.2	44.0*

*Rate difference 1987–1984 = 31.1 (95% confidence interval: 15.3, 46.9).



* Includes endocarditis and sepsis

FIGURE 1—Causes of Death in Montefiore Methadone Program Patients, 1984–87

study period (2.4 to 6.8/1000 person-years). The final category, which also showed a slight increase (0 to 3.4/1000 person-years), was defined as "other," and included deaths from renal failure, pulmonary embolism, bleeding diatheses, stroke, and neoplasms.

Analysis of the HIV serologic status of the subgroup of deceased patients who had been enrolled in the HIV seroprevalence/longitudinal study, as described above, indicated that of nine patients with known serologic status dying from bacterial pneumonia, endocarditis, or sepsis, all were HIV seropositive. In contrast, of 12 patients with known HIV serologic status dying from cirrhosis, overdose, or trauma, 4 (33 percent) were seropositive and 8 (67 percent) seronegative. Of two patients with known serologic status dying of causes classified as "other," both were HIV seropositive. Decedents enrolled in the seroprevalence/longitudinal study did not differ from those in the larger clinic population demographically or by the length of time in methadone treatment.

Incidence of AIDS

The number of incident cases of AIDS newly diagnosed each year increased from five in 1984 to 18 in 1987 (Table 2). The crude annual incidence of AIDS rose from 6.0/1000 in 1984 to 20.4/1000 in 1987. The rate by person-time increased from 5.9/1000 person-years to 20.5/1000 person-years in 1987. In addition to incident cases of AIDS occurring in the treatment program population, 16 additional patients already diagnosed with AIDS were enrolled for treatment during the study period, with a steady increase in such referrals from the first to the fourth year. These patients are excluded from the calculation of AIDS incidence but included in the calculations of hospitalizations and deaths over the four years.

TABLE 2—AIDS Cases among Montefiore Methadone Program Patients, 1984–87

	1984	1985	1986	1987
Midyear Census	828	875	891	882
Person-years	848	860	868	877
Incident cases of AIDS	5	8	11	18
AIDS cases per 1000	6.0	9.1	12.3	20.4
AIDS cases/1000 person-yrs	5.9	9.3	12.7	20.5*
Referred to methadone program after AIDS diagnosis	1	3	5	7

*Incidence rate difference for 1987–1984 = 14.6 (95% confidence interval: 3.8, 25.4).

Hospitalizations

Table 3 presents the frequency of hospitalizations occurring among the Montefiore program patients for different indications during the successive 12-month periods of 1986 and 1987. In 1986, there were 219 hospitalizations, or 202 per 1000 patients; in 1987 there were 269 hospitalizations, or 250 per 1000. A net increase of 71 percent in the frequency of hospitalizations from 1986 to 1987 was observed for AIDS, pneumonia, tuberculosis, and endocarditis/sepsis, which collectively increased from 85 to 145 hospitalizations per 1000 patients over the two years. All other categories showed either minimal increase, no increase, or decrease between the two years.

Discussion

These data document a dramatic threefold increase in overall mortality rates and AIDS incidence and mortality rates from 1984 through 1987, among intravenous drug users in a New York City methadone maintenance treatment program. The parallel increase in deaths from bacterial infections, in patients not diagnosed with AIDS, is consistent with New York City mortality data from the same time period which suggest that such deaths are indeed HIV-related and must be considered in any assessment of the impact of the AIDS epidemic among drug users.[5,7–9] It is noteworthy that among the subgroup of deceased patients with known HIV serologic status, all the bacterial infection deaths occurred in HIV seropositives, compared with only one-third of the deaths due to cirrhosis, overdose, or trauma. These findings are supported by additional data from our prospective studies of HIV infection in the same methadone program population, in which we have observed both a fivefold increased incidence of bacterial pneumonia and an increased death rate from bacterial pneumonia and sepsis in HIV seropositive

TABLE 3—Medical Hospitalizations among Montefiore Methadone Program Patients, 1986–87

	1986 Hospitalizations		1987 Hospitalizations		
Primary Diagnosis	Number n	per 1000*	Number n	per 1000*	Percent Change +/−
AIDS	31	28.6	43	39.9	+40
Pneumonia	50	46.1	87	80.8	+75
Tuberculosis	2	1.9	8	7.4	+302
Endocarditis/sepsis	9	8.3	18	16.7	+101
Suspected endocarditis	6	5.5	6	5.6	+1
Cellulitis/abscess	19	17.5	24	22.3	+27
Trauma	26	24.0	17	15.8	−34
Cirrhosis	8	7.4	7	6.5	−12
Drug overdose	18	16.6	9	8.4	−50
Seizures/stroke	11	10.1	12	11.1	+10
Gastrointestinal bleeding	5	4.6	4	3.7	−20
Cardiovascular/circulatory	10	9.2	6	5.6	−40
Pancreatitis	4	3.7	6	5.6	+51
Other**	20	14.5	22	20.4	+11
Total hospitalizations	219	202.0/1000	269	249.8/1000	+24%

*Total number treated in methadone program in 1986 = 1084 patients, in 1987 = 1077 patients.

**All categories with fewer than five admissions for both years, including: osteomyelitis, urinary tract infection, leg ulcers, diabetes mellitus, cancer, biliary tract disease, asthma, gastroenteritis.

versus seronegative intravenous drug users within the cohort.[21,26]

Previous studies of mortality patterns and rates among intravenous drug users consistently suggested both that death rates among addicts were higher than among the general population and that two-thirds or more of drug user deaths were specifically related to drug overdose, violence or trauma, or alcoholism.[1–4,15–19,27–33] Observed annual death rates in such studies, most of which predated the AIDS era, have ranged between approximately 8 and 16 per 1000.[4,17,18,27,30] Several studies of mortality rates among patients in methadone maintenance programs in the 1970s indicated similar yearly rates, all under 16 per 1000.[16–19] Of note is the fact that the pattern of deaths for the year 1984 in our population was comparable to these historical observations from the pre-AIDS era: the overall level in 1984 was 13.3 per 1000, and close to half of the deaths were due to cirrhosis, overdose, or trauma.

However, by the end of 1987, deaths had increased to 44.2 per 1000, with only 28 percent of deaths due to these causes, and 64 percent of deaths due to AIDS or bacterial infections. Thus, the advent of AIDS has introduced a new risk of alarming magnitude. Also of note regarding causes of death in our study was the small increase in deaths in the "other" category, which included deaths from renal failure and bleeding diatheses, among other causes; in at least two of these cases, the patients were suspected of having HIV-related nephropathy or HIV-related thrombocytopenia, respectively, suggesting that HIV infection may have been implicated in these deaths as well. Further study of larger populations is required to elucidate the relation between HIV infection and less common modes of death such as these; however, it is of interest that New York City vital statistics data indicate a similar increase in such miscellaneous causes of death among drug users over the same interval in which death rates from AIDS and bacterial infections were found to rise more dramatically.[5,8,9]

In addition to the striking increases in mortality among patients in our methadone program population, the increasing incidence of AIDS has generated significant morbidity as well. The rate of 20.4 new cases per 1000 persons in 1987 is an order of magnitude higher than an estimate derived for intravenous drug users in New York in 1983–84.[6] It must also be recognized that our reported incidence is an underestimate of the total number of cases which could now be classified as AIDS, since we intentionally used the more restrictive original CDC AIDS case-definition[23] in order to maintain uniform reporting criteria throughout the study period. While we do not therefore have systematic data regarding the increased number of AIDS cases which would be expected under the revised case-definition criteria,[24] clinical experience involving our patient population has suggested that use of the revised criteria would expand the group of AIDS-diagnosed patients by approximately 30–40 percent (unpublished data). This estimate would be in the same range as what was recently calculated by the CDC concerning the percentage of AIDS cases among intravenous drug users in 1988 meeting only revised case-definition criteria.[34]

For more than 2 percent of all patients in the methadone program population per year to be newly diagnosed with AIDS has had important implications for patient care and clinical practice. HIV infection and AIDS have clearly become the preeminent health problems of our patient population, affecting all areas of treatment, including medicine, nursing, counseling, and administration. Few, if any, other areas of the health care system are now being faced with such a high concentration of patients with or at risk for HIV infection; indeed, drug treatment programs may be among the only clinical settings in which the entire patient population is defined by a history of high-risk behavior for HIV infection. Observations that 50 percent or more of intravenous drug users in New York are already infected with HIV,[35,36] and data from a 1985 seroprevalence study in our own population indicating close to a 40 percent prevalence of HIV antibody,[20–22] suggest that any such treatment program for drug users cannot help but be transformed by the consequent impact of AIDS on its patient population. This trend has been amplified by the acceptance into drug treatment of new patients from hospital AIDS units, where fewer than 30 percent of drug users newly diagnosed with AIDS have been found to be enrolled in drug treatment programs.[37]

The need to expand the availability of drug treatment in response to the AIDS epidemic has been stressed repeatedly.[7,12,38–42] However, our data suggest that in order to deal effectively with the AIDS crisis, treatment programs will need to receive much-expanded support for on-site medical services and ancillary care to address the steadily increasing burden of HIV-related morbidity and mortality among their patient populations.

Treatment programs are indeed well-situated to provide comprehensive care to patients with HIV infection, including counseling, family supportive services, and, increasingly, primary medical care interventions such as antituberculous chemotherapy,[22] zidovudine, aerosolized pentamidine, and reproductive health care services for at-risk women. However, the growing volume of methadone treatment program patients with AIDS and HIV-related conditions threatens to overwhelm existing drug treatment resources unless structural changes are made to address the new dimensions of these patients' need for care. Such changes should begin with a thorough re-examination of the character and mission of drug treatment programs, including funding, reimbursement mechanisms, staff training and development, need for interdisciplinary collaboration, and the relationship between drug abuse treatment agencies and local and state health departments. Failure to act now may result in a missed opportunity to use a key point of access to intravenous drug users at the center of the expanding AIDS epidemic, and (because of the great stresses which AIDS places on drug treatment program staff) may also lead to the deterioration of existing services.

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ANA Issues Call for Abstracts of Computer Demonstrations in Nursing

The American Nurses' Association (ANA) Council on Computer Applications in Nursing will sponsor a computer demonstration theater and software exchange at the ANA Biennial Convention in Boston, June 17–19, 1990. The council is now issuing a call for abstracts of demonstrations in computer applications in nursing. All types of nursing software and related hardware and peripherals are being solicited.

The deadline is November 15, 1989 for submission of written abstracts of demonstration requests. Individuals submitting abstracts will be notified of acceptance by December 15, 1989. Attendees are urged to bring software they would like to exchange; diskettes should be formatted using MS-DOS. Several public domain software packages will be available for review and copying. A limited number of blank diskettes will be available on-site for purchase

To obtain special rules on abstract submission as well as information on hardware to be used, contact: Catherine Taylor Foster, PhD, RN, ANA, 2420 Pershing Road, Kansas City, MO 64108. Tel: (816) 474-5720, ext. 326.

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Suicide Risk among Women with Alcohol Problems

Edith S. Lisansky Gomberg, PhD

Abstract: The current study compares 301 alcoholic women, ages 20 to 50, interviewed in 21 treatment facilities, and a control group of nonalcoholic women matched for age and socioeconomic status of family of origin. A significantly higher percentage of alcoholic women (40.0% vs 8.8%) reported having made suicide attempts, a difference of 31.2 percentage points (95% CI = 23.9, 38.5). Age comparisons within the alcoholic sample show the percentage of younger alcoholic women attempting suicide to be twice as great as the percentage of the alcoholic women suicide attempters in their forties. Such age differences were not found among the nonalcoholic control group. The findings suggest that youthfulness and alcohol/drug abuse are the critical combination for high risk. Awareness of the suicide attempt risk potential is necessary both for emergency room personnel and for substance abuse facility workers. (*Am J Public Health* 1989; 79:1363–1365.)

Introduction

Almost a century ago, Durkheim's classic work on suicide pointed out that more men than women committed suicide, but that suicide attempts were more frequent among women.[1] There have been attempts to explain this gender difference in terms of sex roles and cultural attitudes.[2–4] Durkheim also noted the relation between suicide and alcoholism and, indeed, suicide is one of the more frequent causes of death among alcoholics.[5–8] This relationship between alcoholism and suicide has been related to depression and other psychiatric disorders, to erosion of social networks, and to disrupted interpersonal relationships.[9–12]

Similar male:female patterns of completed suicide and suicide attempts seem to prevail among alcoholic persons.[13–16] Goodwin concluded that there was an association between alcoholism and suicide among White male alcoholics during the middle years of life, whereas, ". . . attempters generally are younger and are more likely to be women . . ."[7]

A review of the epidemiology of suicide attempts[17] shows the risk years to be the twenties and the same review showed more female than male attempters in the countries and time periods studied. A recent analysis of male and female suicide rates[18] found completed suicide rates are significantly higher for men.

Suicide rates are high among both male and female alcoholics. A recent study of predictors of mortality among women alcoholics found significantly higher mortality for alcoholic women than for the general population of women; "accidents and violence" accounted for 26 percent of the deaths, ranking second as the cause of death in the alcoholic sample.[19] There are few studies of suicide attempts by alcoholics, by women alcoholics, or by women alcoholics of different ages. We studied suicide attempts in alcoholic and nonalcoholic women by age group.

Methods

Three hundred and one White women, admitted to 21 alcoholism treatment facilities in Michigan, completed a one-to-two-week period of detoxification and were then interviewed by trained female interviewers of the Field Office, Institute for Social Research, University of Michigan.

The 21 facilities included inpatient, outpatient, small town, and urban facilities, and the women were referred to the facility by self, physician, family member, the court, friends, etc. All consecutive admissions between the ages of 20 and 50 were asked to participate; refusal rate was 7 percent. All respondents were White because available evidence indicates differences in age, drinking patterns, and alcohol-related problems when White and Black alcoholic women are compared.[20] Age at onset was, inevitably, related to current chronological age, i.e., alcoholic women in their twenties had a mean age at onset of 19.58 years (standard deviation = 3.18); alcoholic women in their thirties reported their mean age at onset as 28.34 years (standard deviation = 5.35); and the alcoholic women in their forties reported mean age at onset as 36.52 years (standard deviation = 5.93).

When 200 of the 301 women in the alcoholic sample had been interviewed, the demographic information was evaluated in order to obtain a nonalcoholic sample of women approximately matched on socioeconomic status. The control women were White, between 20 and 50 years of age, and approximately of the same social class as the alcoholic women. They were selected by nomination; the alcoholic women who remained to be interviewed (100) and women in the local chapters of Alcoholics Anonymous and Women in Sobriety were asked to give the name of an acquaintance or neighbor who was *not* alcoholic. Refusal rate of the nominated women was the same as that of women in treatment; when funding was terminated, interviews had been completed with 34 nonalcoholic women in their twenties, 53 in their thirties, and 50 in their forties. Of these 137 control women, none had ever needed a drink before breakfast and none have ever sought help because of their drinking, three had gotten into trouble at school, one had been arrested for driving under the influence, and one reported that she had "neglected obligations" for two days. Two said that they had once "wanted to stop drinking but couldn't." As a group, however, they were nonalcoholic.

The economic status of family of origin and the white/blue collar status of respondents' parents were similar in the alcoholic and nonalcoholic groups.[21] The control group was also compared with a random sample of women interviewed in an earlier, national survey.[22] When women between the ages of 20 and 50 from the national sample were compared with the present control group on items dealing with psychiatric symptomatology, no significant differences were apparent.*

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*Data available on request to author.

The standardized interview schedule include items dealing with demographics; early family experience; drinking history; marital, parenting and employment experience; health; social supports; pregnancies; drug usage; and psychiatric symptomatology. The interview was composed of items culled from several alcoholism study interviews in the Connors Collection of the Rutgers Center of Alcohol Studies, from the Diagnostic Interview Schedule (DIS) of the National Institute on Mental Health, from the Center on Epidemiological Studies Depression Scale (CES-D), and from several earlier surveys conducted at the Institute for Social Research. After field-testing and revising, the interview schedule was reviewed by a panel of three clinicians and a panel of three recovered alcoholic women.[21]

Although alcoholic and nonalcoholic women were matched for age and for socioeconomic status of family of origin, the alcoholic women reported more behavioral, emotional and impulse control problems in childhood and adolescence.[21,23] There are significant differences between the two groups of women in educational achievement, marital history and marital status, and occupational level.[21]

Results

The difference between alcoholic and nonalcoholic women in the proportion reporting any suicide attempt is striking: 40.0% versus 8.8%, a difference of 31.2 percentage points (95% CI = 23.7, 38.5) (Table 1).

TABLE 1—Proportions of Alcoholic and Nonalcoholic Women Reporting Suicide Attempts, by Age

Age (years)	Alcoholic N/Total (%)	Nonalcoholic N/Total (%)	% Difference	95% Confidence Interval
20–29	50/99 (50.5)	3/34 (8.8)	41.7	27.8, 55.5
30–39	46/108(43.0)	5/53 (9.4)	33.6	21.2, 45.9
40–49	24/94 (25.5)	4/50 (8.0)	17.5	5.9, 29.2
All Women	120/301(40.0)	12/137(8.8)	31.2	23.9, 36.5

Among alcoholic women, suicide attempts were twice as common at ages 20–29 as at ages 40–49. The same age differences, however, did not appear in the nonalcoholic sample. These results do not support the idea that younger women in general are at more risk for suicide attempts than women of other ages.

When the women were interviewed, they were asked about the use of substances other than alcohol, and the alcoholic women reported significantly more often that they had used or were currently using nicotine, prescribed drugs, and banned substances (Table 2). Those who had made suicide attempts were also more likely to have used any of the drug substances asked about. There were too few nonalcoholic women who reported a suicide attempt (N = 12) for meaningful comparisons; it is of interest that those who have made a suicide attempt are also more likely to be users of Valium and sedatives.

Among alcoholic women, there was a clear difference in the reported psychiatric symptoms of suicide attempters and nonattempters. Those alcoholic women who have made suicide attempts also reported significantly more tension, indecisiveness, anxiety and nervousness (Table 2). The report on psychosomatic symptoms was mixed: attempters report more dizziness, hand trembling, and headaches than nonattempters but there is little difference between the two groups in loss of appetite, upset stomach, shortness of breath, hives, or sweating hands.

There was a positive but not strong relation between paternal alcoholism and suicide attempts. Of the alcoholic women who made at least one suicide attempt, 63 percent reported a problem-drinking father; among the nonattempters it was 48 percent. Among the nonalcoholic women, 73 percent of attempters reported having had a problem-drinking father; among nonattempters, it was 28 percent. Among the alcoholic women having an older sibling who was a heavy or problem drinker while the respondent was growing up differentiated between those who made at least one suicide attempt and those who did not: 66 percent and 52 percent. It is of interest that maternal alcoholism did not differentiate

TABLE 2—Ever or Current Drug Use and Psychiatric Symptoms Reported by Alcoholic Women, Suicide Attempters and Nonattempters

Drug/Symptoms	Attempters N/Total (%)	Nonattempters N/Total (%)	Difference	95% Confidence Intervals
Drug				
Nicotine	107/120 (89.2)	140/180 (77.8)	11.4	3.1, 19.7
Valium	93/120 (77.5)	106/180 (58.9)	18.6	8.2, 29.2
Stimulants	86/119 (72.3)	91/180 (50.6)	21.7	10.8, 32.6
Sedatives	79/120 (65.8)	74/180 (41.1)	24.7	13.5, 35.9
Marijuana	82/120 (68.3)	77/180 (42.8)	25.5	14.5, 36.6
Psychodelics	54/120 (45.0)	45/180 (25.0)	20.0	9.0, 31.0
Cocaine	51/120 (42.5)	37/180 (20.6)	21.9	11.3, 32.6
Heroin	17/120 (14.2)	7/180 (3.9)	10.3	3.4, 17.2
Symptom				
Feel explosive	59/119 (49.6)	40/178 (22.5)	27.1	16.2, 38.0
Indecisive	51/120 (42.5)	37/179 (20.7)	21.8	11.1, 32.5
Tense and nervous	43/120 (35.8)	31/180 (17.2)	18.6	8.4, 28.8
Fearful, anxious	31/120 (25.8)	22/179 (12.3)	13.5	4.3, 22.8
Difficulty concentrating	38/119 (31.9)	24/179 (13.4)	18.5	8.7, 28.3
Difficulty getting up in the morning	34/120 (28.3)	26/178 (14.6)	13.7	4.1, 23.4
Thoughts of others harming you	34/120 (28.3)	27/180 (15.0)	13.3	3.7, 23.0
Thoughts of harming other people	37/120 (30.8)	26/180 (14.4)	16.4	6.6, 26.1

between suicide attempters and nonattempters, alcoholic or controls.

Discussion

The results suggest that it may not be youthfulness per se which is critical in the relatively high percentage of young female suicide attempters seen in hospital emergency rooms but the combination of youth and substance abuse. Among the nonalcoholic women in this study, there was little difference in the proportion of suicide attempters among younger and older women.

An attempt was made to deal with the question of bias in the selection of clinical subjects by interviewing the patients of 21 different treatment facilities. While the facilities were limited to southeastern Michigan, they represented a wide range of treatment orientations and methods. One may question whether alcoholic and control women are equally likely to admit to a suicide attempt, to remember such an attempt, or to report a suicide attempt. There is no unequivocal answer to this question, but if experimental procedures are reasonably well planned, and the interviewers skilled so as to establish excellent rapport, one can assume that the groups of women were reasonably comparable on this score.

Why the age differential among alcoholic women? One could speculate about the role of depression, impulsivity, hostility, hysterical trends, risk-taking behaviors, and combinations thereof—and the linkage between such patterns of behavior and heavy alcohol intake among different age groups of alcoholic women. Analyses of other data obtained in this study of alcoholic women[21] show nonsignificant age differences in depression among alcoholic women, but significant age differences in early history of impulse control.[23]

Emergency room personnel are acutely aware of the problems of drug overdose and drug abuse. It is worth reiterating: while it is true that young women are a high-risk group for suicide attempts,[24] it is also true that such attempters are likely to have a history of alcohol or other drug abuse. Whether this is due to ". . . social stigmatization and disapprobation"[25] is beside the point. A person presenting with a suicide attempt is in need of assessment and appropriate referral from the emergency room. As for substance abuse treatment facilities, it is important that they be aware of the suicide attempt potential of young women drug abusers; the abuse of a given substance or substances may not be preventable at the point of entry into treatment but suicide attempts should be investigated with the goal of prevention of such attempts.

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Prevention '90 Set for April 18–22, 1990 in Atlanta

"Assuring the Public's Health" will be the overall theme of the Prevention 90 meeting, to be held April 18–22, 1990 in Atlanta. This will be the 7th annual national meeting on disease prevention and health promotion sponsored by the American College of Preventive Medicine, the Association of Teachers of Preventive Medicine, and co-sponsored by a number of other health organizations, including the American Public Health Association.

To receive further information and obtain the standard abstract forms, contact: Joanne Angle, Association of Teachers of Preventive Medicine, 1030 15th Street, NW, Suite 410, Washington, DC 20005. Telephone: (202) 789-2928.

The deadline for abstract submission is October 6.

Business Recession, Alcohol Consumption, Drinking and Driving Laws: Impact on Oklahoma Motor Vehicle Fatalities and Fatal Crashes

ANDREAS MULLER, PHD

Abstract: In 1982, Oklahoma enacted a series of drinking and driving laws. In the ensuing years, motor vehicle fatalities and fatal crashes were reduced by one-third. Factors contributing to this reduction were examined using interrupted time series analysis of monthly rates of motor vehicle deaths and fatal crashes for the period January 1980 to December 1986. Decreasing per capita alcohol consumption and increased unemployment apparently account for most of the fatality and fatal crash reduction in Oklahoma. The enactment of two traffic safety laws—one specifying the illegal blood alcohol concentration level (BAC law) and the other facilitating license withdrawal from suspected drunk drivers (administrative per se law)—together reduced Oklahoma traffic deaths and fatal crashes by about 9 percent. The effectiveness of the laws appeared to be greatest in the first two years following their enactment. (*Am J Public Health* 1989; 79:1366–1370.)

Introduction

In 1983, Oklahoma motor vehicle fatalities decreased by more than one-fifth and have continued to decrease since then.[1] The oil shortage and the reduction of the national speed limit in 1973 had less impact on motor vehicle fatalities. The only comparable decrease in such deaths occurred in 1942, reflecting the American entry into World War II.

In July 1982, the Oklahoma state legislature established a blood alcohol concentration level of .1 g/dl (22 mmol/L) as "evidence per se" of driving while intoxicated (BAC law). In April 1983, this law was reinforced by an administrative per se law (ADM law) permitting automatic license revocation, if a police officer was refused the right to administer a blood alcohol test to a suspected drunk driver. In September of the same year, Oklahoma raised the minimum age for purchase, possession, and consumption of beverages in excess of 3.2 percent alcohol concentration from 18 to 21 years.[2] An interrupted time series analysis of monthly motor vehicle fatalities and fatal crashes from January 1975 to March 1986 suggested that the law reduced Oklahoma fatalities and fatal crashes by about one-third.[3] In addition, the effectiveness of the administrative per se law appeared to increase over time; this finding departed from previous studies showing the deterrent effect of drunken-driving laws and related enforcement campaigns to be short-lived, reviewed elsewhere.[4]

The findings of an effect of the Oklahoma laws contrast with studies of similar legislation enacted in California, Maine, and Massachusetts conducted by Hilton[5] and Hingson, *et al*,[6] which have found the trends in monthly single-vehicle night time fatal crashes comparable to those in adjacent states without the legislation and to trends in non-alcohol-related fatal crashes.

A recent nationwide study by the Insurance Institute for Highway Safety (IIHS) for the period 1978–85 found that administrative per se laws and BAC laws reduced fatal crashes by 4.6 percent and 2.4 percent, respectively. State-specific variation in effectiveness may occur and can only be determined by additional analyses.

Unlike other parts of the United States, Oklahoma experienced an oil-related economic boon during the period 1980–82 followed by a severe recession beginning with Penn-Square Bank failure in July 1982. The recession was prolonged by a sharp drop in crude oil prices in January 1986. The unemployment rate for the civilian labor force in Oklahoma increased from a 4–5 percent level in 1980–81 to over 10 percent in early 1983, receded to 7–8 percent in 1984–85, and increased again in 1986 (Figure 1). Business conditions affect the motor vehicle fatality trend since unemployment reduces work-related travel and disposable income, making alcohol consumption and leisure travel less likely.[8]

Apart from the effects of business cycles, alcohol consumption patterns have changed over recent years, with decreased consumption of distilled liquor and increased consumption of beer and wine having the overall effect of reducing the per capita amount of pure alcohol (ethanol) consumption since 1982.[9] Correspondingly, between 1982 and 1986, the proportion of fatal crashes involving intoxicated drivers has decreased by 11 percent.[10] Per capita pure alcohol consumption by Oklahomans (Figure 2) parallels the national trend showing a gradually decreasing trend since 1982.

Since alcohol consumption contributes to motor vehicle crashes and deaths, the gradual reduction in alcohol consumption in Oklahoma should have resulted in fewer alcohol-related crashes regardless of the imposition of stricter alcohol-related traffic safety laws. The effect of such legislation would be overestimated if the alcohol consumption trend and the effect of the business recession were not controlled.

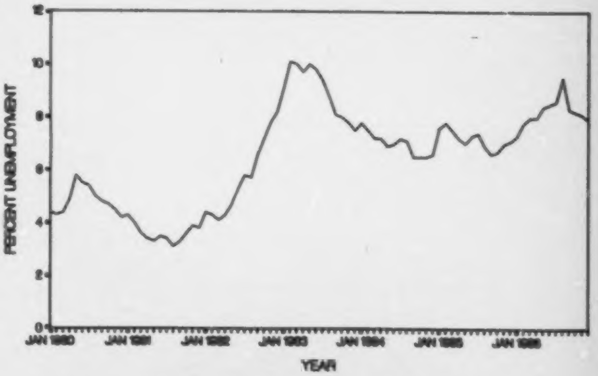

FIGURE 1—Monthly Unemployment Rate, Oklahoma 1980–86
SOURCE: Oklahoma Employment Security Division

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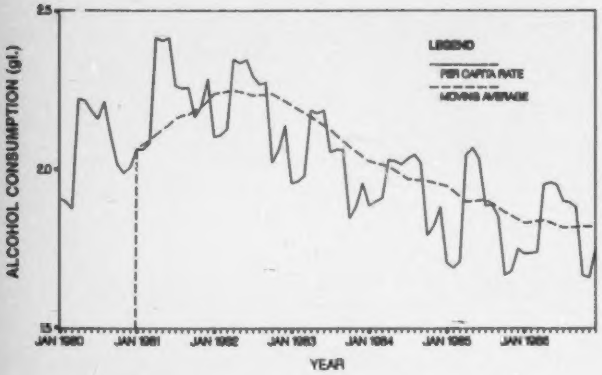

FIGURE 2—Monthly per capita Alcohol Consumption and Moving Averages, Oklahoma 1980–86
SOURCE: Appendix A

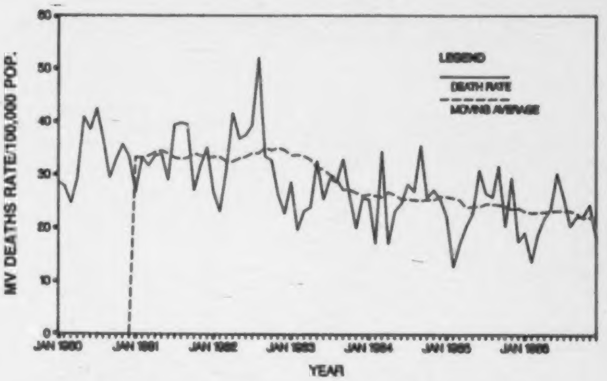

FIGURE 3—Monthly Motor Vehicle Death Rates and Moving Averages, Oklahoma 1980–86
SOURCE: Department of Transportation, Oklahoma Highway Safety Office

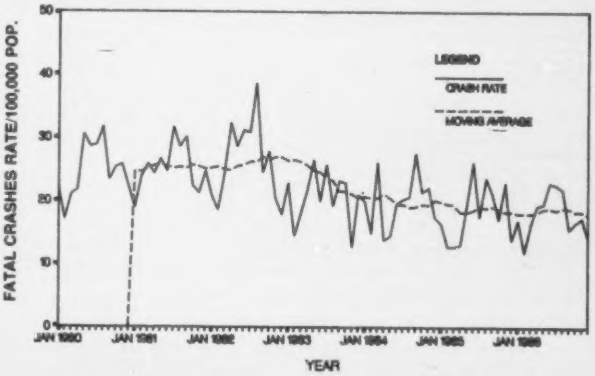

FIGURE 4—Monthly Fatal Motor Vehicle Crash Rates and Moving Averages, Oklahoma 1980–86
SOURCE: Department of Transportation, Oklahoma Highway Safety Office

Methods

The time series analysis was based on three sets of data pertaining to motor vehicle fatalities and fatal crashes for the period January 1980 to December 1986 (n = 84). The first data set included all deaths whose cause was coded E810-E825, ICDA-9 for Oklahoma residents.[11] About 3 percent of the fatalities occurred beyond state boundaries.

The second data set pertains to the number of crashes involving at least one fatally injured person. Fatal crashes involving heavy trucks and buses (7 percent) were deleted, since operators of those vehicles are likely to be out-of-state residents unfamiliar with Oklahoma's laws regarding drinking and driving.

The third data set is a subset of the second, removing fatal crashes involving drivers under 21 years of age. This deletion controls for the potentially confounding effect of the change in the minimum drinking age law. Due to the small number of fatal crashes involving persons under 21 years of age, no reliable analysis of the effect of the minimum drinking age law could be performed.

To control for changes in Oklahoma's population, we divided the time series by the appropriate population estimates.[12] The time series of fatal crashes involving drivers over 20 years of age was divided by the corresponding age-specific population estimates; the two remaining series were divided by estimates of the total population. The procedure resulted in three sets of rates:

- monthly motor vehicle deaths per 100,000 population,
- monthly fatal crashes per 100,000 population, and
- monthly fatal crashes involving adult drivers per 100,000 population over 20 years of age.

The data pertaining to fatal crashes were extracted from Department of Transportation files and provided by staff of the Oklahoma Highway Safety Office. The unemployment data including estimates for 1986 were obtained from staff of the Oklahoma Employment Security Division.

The Oklahoma Department of Public Safety released license revocation data pertaining to BAC testing. Inspection of the time series and discussion with the administrator of the records division showed that reporting lags of one to two months occurred occasionally. To even out such lags, the data were averaged by quarter.

Oklahoma per capita alcohol consumption was estimated from data provided by the alcoholic beverage industry (Appendix A). The time series were analyzed by ordinary least squares multiple regression using SPSS-X Trends computer software[13] (Appendix B).

Results

Monthly time series of motor vehicle death rates and fatal crash rates in Oklahoma (Figures 3, 4) showed typical seasonality with the highest rates in the summer months and the lowest rates in February. The time series for the subgroup of fatal crashes with adult drivers (not shown) was very similar to those shown. All time series have one exceptionally high rate, August 1982, an outlier removed in subsequent analyses (see Appendix B). The trend began to decrease late in 1982, reached a new plateau by the last quarter of 1983, and decreased more slowly thereafter.

Table 1 shows the best fitting regression models using dummy variables and counters for the three monthly time series. The models indicate that with the enactment of the BAC law (July 1982) the level of the time series dropped between 5.9 and 12.2 percent. With the enactment of the administrative per se law (April 1983), the levels of the time series further dropped between 9.7 and 13.2 percent when compared with levels before the enactment of the law. The

TABLE 1—Effects of Blood Alcohol Concentration and Administrative per se Laws on Motor Vehicle Deaths and Fatal Crashes, Oklahoma 1980–86

Independent Variables		MV Death Rate (100,000 Pop)	MV Fatal Crash Rate (100,000 Pop)	MV Fatal Crash Rate Adult Drivers (100,000 Pop >20 yrs)
BAC Law	b	−4.05	−2.34	−1.87
	s.e.	1.49	1.32	1.68
	p	.01	.08	.27
ADM Law	b	−3.78	−2.44	−4.21
	s.e.	1.77	1.57	1.99
	p	.04	.13	.04
ADM Law counter	b	−.099	−.078	−.093
	s.e.	.044	.039	.05
	p	.03	.05	.07
F		14.4	8.8	8.9
Adj. R^2		.69	.57	.57
Durbin-Watson		1.87	2.09	1.86
Mean rate before laws		33.2	25.3	32.0
Estimated % Reduction Due to Laws				
BAC Law		−12.2	−9.3	−5.9
ADM Law		−11.4	−9.7	−13.2
ADM Law counter		−13.4	−13.8	−13.1
Total		−37.0	−32.8	−32.2

The models omit coefficients for intercept and 11 dummy variables representing seasonality.

p = probability of two-tailed test; b = 0.

n = 84

TABLE 2—Effects of Administrative per se Law, Per Capita Alcohol Consumption and Unemployment Rate on Motor Vehicle Deaths and Fatal Crashes, Oklahoma 1980–86

Independent Variables		MV Death Rate (100,000 Pop)	MV Fatal Crash Rate (100,000 Pop)	MV Fatal Crash Rate Adult Drivers (100,000 Pop >20 yrs)
Alcohol consumption	b	11.42	10.74	17.52
	s.e.	5.46	4.64	6.25
	p	.04	.02	.01
Unemployment rate	b	−.78	−.31	−.25
	s.e.	.33	.28	.38
	p	.02	.28	.52
ADM law	b	−3.53	−2.58	−2.27
	s.e.	1.88	1.60	2.15
	p	.07	.11	.29
F		10.9	9.0	7.4
Adj. R^2		.63	.57	.52
Durbin-Watson		2.00	2.14	2.05
Mean rate before laws		33.2	25.3	32.0
		Estimated % Reduction due to Law		
ADM Law		−10.6	−10.2	−7.1

The models omit coefficients for intercept and 11 dummy variables representing seasonality.

p = probability of two-tailed test; b = 0.

n = 84

negative coefficients for the ADM time counter indicate that the rates continued to decrease. By December 1986, the fatality and fatal crash rates were about one-third lower than before the enactment of the laws.

Table 2 shows the decrease in motor vehicle deaths and fatal crashes associated with increased unemployment, reduced alcohol consumption, and the administrative per se law. Depending upon the time series, the law appeared to have reduced fatality and fatal crash rates between 7.1 and 10.6 percent. However, the coefficients for the BAC law were inconsistent in sign and statistically insignificant. Therefore, the BAC law dummy variable was deleted from the final mode specification.

Another analysis, shown in Table 3, includes fatal crashes in Oklahoma which police determined not to be alcohol related. This series is a proxy for changes in extraneous factors, such as travel or weather. In addition, the dummy variables and counters representing BAC and administrative per se laws are replaced by a measure of enforcement of drunken driving: the number of licenses revoked involving BAC tests.

The models fit the time series very well, mainly because the series is regressed on the time series of nonalcohol-related fatal crashes. The multiple regression estimates show that per capita alcohol consumption increases fatality and fatal crash rates, while increased law enforcement has the opposite effect. Increased enforcement reduced motor vehicle death rates by 9.6 percent after the enactment of the administrative per se law, and by 5.0 percent during the nine-month period (7/82 to 3/83) during which only the BAC law was in effect. The estimates are similar for the time series of fatal crash rates.

In Table 3 the unemployment rate showed a weak direct effect, because part of the overall unemployment effect was shared by the trend of nonalcohol-related crashes.

Police enforcement varied over the study period (Figure 5), with a gradual increase in license revocations following enactment of the BAC law. Enforcement was highest during the first quarter after the enactment of the administrative per se law. Thereafter, enforcement activity declined until early 1985 despite some seasonal variation. Thus, the effect of the two laws should have been most pronounced during the first year after enactment of the administrative per se law. It is estimated that fatal crash rates of adult drivers were reduced by 12.3 percent during the first year of enactment of the administrative per se law, 10.8 percent during the second year of enactment, and 7.8 percent during the remaining 21 months (4/85–12/86). Since both laws were in effect from April 1983 on, the estimates reflect the combined effective-

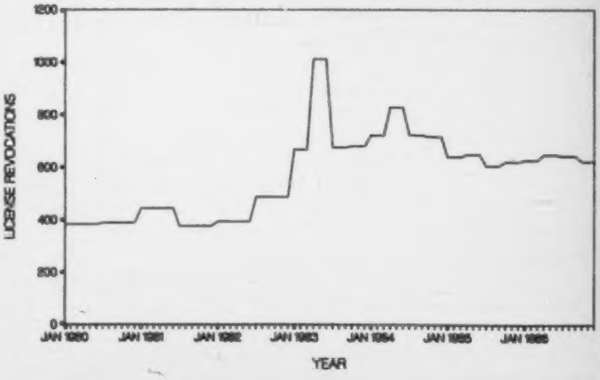

FIGURE 5—License Revocations due to BAC Tests, Oklahoma 1980–86
NOTE: Data represent quarterly averages
SOURCE: Oklahoma Department of Public Safety

TABLE 3—Effects of License Revocation, Per Capita Alcohol Consumption, Unemployment Rate and Nonalcohol-related Crashes on Motor Vehicle Deaths and Fatal Crashes, Oklahoma 1980–86

Independent Variables		MV Death Rate (100,000 Pop)	MV Fatal Crash Rate (100,000 Pop)	MV Fatal Crash Rate Adult Drivers (100,000 Pop >20 yrs)
Non-alcohol-related fatal crash rate	b	1.23	1.20	1.19
	s.e.	.16	.09	.09
	p	<.001	<.001	<.001
Alcohol consumption	b	10.57	7.28	10.97
	s.e.	2.91	1.77	2.23
	p	<.001	<.001	<.001
Unemployment rate	b	−.24	.14	.18
	s.e.	.32	.20	.26
	p	.47	.49	.50
License revocations	b	−.0107	−.0091	−.0104
	s.e.	.004	.002	.003
	p	007	<.001	.001
F		23.0	38.4	35.8
Adj. R^2		.80	.87	.86
Durbin-Watson		1.87	1.98	2.03
Mean rate before laws		33.2	25.3	32.0
Estimated % reduction due to Increased Law Enforcement				
BAC Law (7/82–3/83)		−5.0	−5.6	−5.1
ADM + BAC Law				
(4/83–3/84)		−12.1	−13.6	−12.3
(4/84–3/85)		−10.6	−11.9	−10.8
(4/85–12/86)		−7.6	−8.5	−7.8
(4/83–12/86)		−9.6	−10.8	−9.8

The models omit coefficients for intercept and 11 dummy variables representing seasonality.

p = probability of two-tailed test; b = 0.

n = 84

ness of the laws. The effectiveness of the BAC law for the nine-month period preceding the introduction of the administrative per se law is estimated at about 5 percent.

Discussion

The enactment of a BAC law and an administrative per se law in Oklahoma reduced fatality rates and fatal crash rates by about 9 percent in the four years following their enactment. This estimate is less than one-third of the size found in a previous study of the effectiveness of the Oklahoma administrative per se law.[3] The difference in estimates is due mainly to the control of alcohol and to unemployment trends which were not considered in the earlier study.

The effect of the BAC law was not statistically significant when other variables were controlled and deleted from the analysis (Table 2). The most plausible reason is that the enactment of the BAC law nearly coincided with rapidly rising unemployment making the BAC regression estimate unreliable. However, when license revocations were used to determine the effectiveness of the laws, the BAC law was estimated to have reduced fatality and fatal crash rates by about 5 percent. This estimate pertains to the initial nine-month period separating the two laws; the long-term effectiveness of this legislation may be different. A recent nationwide study estimated an average (long-term) effect of 2.4 percent for BAC laws and 4.6 percent for administrative per se laws.[7] These estimates approximate the combined long-term effect (7.2 percent), past two years, found in this study.

Deleting fatal crashes involving drivers under 21 years of age did not markedly affect the estimated effectiveness of the laws (Table 3). This finding suggests that the BAC and the administrative per se law are responsible for the decrease in fatal crash rates rather than the raising of the minimum drinking age law.

The accuracy of police-reported alcohol-impaired driving is a concern in this study. Oklahoma police could not determine alcohol involvement for 11 percent of drivers involved in fatal crashes. The percentage was about the same from 1980 to 1986 suggesting no increased effort by police to report alcohol involvement in fatal crashes.

In addition, an increase in the reporting of nonalcohol-related fatal crashes coinciding with the enactment of the laws could not be found. Such a change would be expected, if police officers became more lenient when faced with administering harsher legal sanctions. Errors in the reporting of alcohol involvement would not bias the regression estimates unless the error rate changed over time; no evidence supporting this conclusion could be found.

The gradual reduction in per capita alcohol consumption has had a substantial effect on Oklahoma's fatal crash trend. The annual per capita consumption dropped by about .4 gallons pure alcohol between 1981 and 1986. The corresponding decrease in motor vehicle death and fatal crash rates is estimated at 12–14 percent, controlling for changes in unemployment and drinking and driving laws.

The total effect of the business recession, measured by the unemployment rate, is less clear. Since the unemployment rate was on the average 4 percent higher after the enactment of the drinking and driving laws, about 10 percent of the reduction in death rates should be due to increased unemployment.

However, increased unemployment appears to have had little direct effect on fatal crash rates, although indirect effects via the other variables are consistent with the findings. For instance, unemployment had a significant negative effect on nonalcohol-related fatal crashes. A 4 percent increase in the unemployment rate reduced fatal crashes by 2/100,000 controlling for seasonal variations. Thus, increased unemployment, by reducing nonalcohol-related fatal crashes, could have contributed to a 7 percent reduction in the fatal crash rates.

APPENDIX A

Per Capita Alcohol Consumption Index

Data for estimating monthly per capita consumption of pure alcohol (ethanol) in Oklahoma are provided by the Distilled Spirits Council of the United States, the Beer Institute and the Wine Institute. The data pertain to shipment and sales records regularly compiled by the alcoholic beverage industry. Beer and wine data were provided by month, while distilled spirits data could only be obtained by year. To estimate monthly pure alcohol consumption, the data were converted into gallons and the annual amount of distilled spirits was divided by 12. The following formula was used to derive the estimated monthly per capita alcohol consumption.

$$\text{P.C. ethanol consumption} = (.414 * \text{DSP} + .033 * \text{Beer} + .129 * \text{Wine}) * 12/\text{POP}; \quad (1)$$

where: DSP = distilled spirits (gl.);

POP = Oklahoma resident population over age 20;

The coefficients for distilled spirits and wine refer to the average alcohol concentration estimated by the Alcohol Epidemiological Data System.[14] The average alcohol concentration of beer is less in Oklahoma than in other states, due to the mandated 3.2 percent limit for beer sold in grocery and convenience stores. According to Oklahoma tax receipt data, only 5 percent of beer has higher alcohol concentration. The estimated average alcohol concentration of beer sold in Oklahoma is estimated to be 3.3 percent.

APPENDIX B

Statistical Model and Method of Analysis

The time series are analyzed by a form of multiple regression which uses dummy variables and dummy counters to represent changes in the level (mean) and trend (slope) of the time series.[15,16] Another set of dummy variables is used to represent seasonality.[17] The basic model for this study is as follows:

$$Y_t = a + b_1X_1 + B_2X_2 + B_3X_3 + B_4X_4 + \Sigma B_iX_i + e_i; \qquad (2)$$

where: Y_t = monthly motor vehicle death rate (fatal crash rate);
a = reference category;
b_i = regression coefficients;
X_1 = linear trend counter, coded 1 to 84;
X_2 = dummy variable, coded '0' before BAC law went into effect and '1' thereafter;
X_3 = dummy variable, coded '0' before administrative per se law went into effect and '1' thereafter;
X_4 = dummy variable counter, coded '0' before administrative per se law went into effect and '1' to n thereafter'
ΣX_i = set of 11 dummy variables identifying months (January to November) representing seasonality;
e_t = error term;

The set of monthly dummy variables controls for seasonal fluctuations in the time series, while the linear trend counter X_1 controls for any existing secular, linear trend which is independent of the effects of traffic safety laws. The effects of the two laws are modeled by variables X_2 to X_4. The laws may be effective, if one or more of the coefficients associated with variables X_2 to X_4 are negative and statistically significant. To prevent multicollinearity, a dummy variable counter for the BAC law was excluded from the model.

In order to control for unemployment and alcohol consumption trends, these variables were added to equation (2). The best-fitting model was determined by deleting statistically insignificant terms and by inspecting residual plots.

First, extreme outliers ($z = \pm 2.57$ standard deviations) were replaced by linear interpolation using data pertaining to adjacent months. Data for August 1982 and March 1984 (observations 32 and 51) were replaced by this method normalizing the distributions of residual plots.

Second, the distribution of residuals for the best-fitting models did not significantly depart from homoscedasticity, although the variances of residuals were somewhat smaller for the period after the laws went into effect. Converting the dependent variables to their natural logarithms further stabilized the variance of residuals, but did not change the regression estimates substantially. The time series were also analyzed by weighted least squares regression without substantial change in findings.

Third, besides Durbin-Watson tests of the first order autocorrelation, the residuals were tested by the Box-Ljung statistic. The latter tests showed that higher order autocorrelations were small and consistent with a random process. The autocorrelation plots indicated no significant spikes at critical lags for models including the control variables.

ACKNOWLEDGMENTS

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6th National Cancer Communications Conference Set for January 1990

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Six-Year Follow-up of the First Waterloo School Smoking Prevention Trial

BRIAN R. FLAY, DPHIL, DAVID KOEPKE, PHD, SHIRLEY J. THOMSON, MA, SUSANNE SANTI, MSC, J. ALLAN BEST, PHD,
AND K. STEPHEN BROWN, PHD

Abstract: This paper reports six-year follow-up data from the first large-scale randomized trial of the social influences approach to smoking prevention. In 1979, 22 schools were randomly assigned to program or control conditions. Students in program schools received a social influences curriculum in six core and two maintenance sessions in grade 6, two booster sessions in grade 7, and one booster session in grade 8. All students were assessed at pretest (T1), immediate posttest (T2), end of grade 6 (T3), beginning and end of grade 7 (T4 and T5), end of grade 8 (T6), and grades 11 and 12 (T7 and T8). Ninety percent of study students were relocated and data obtained from over 80 percent of them at T8. Program effects on experimental smoking observed in grades 7 and 8 had completely decayed by T8, six years after the beginning of the program. Grade 6 smoking experience and social risk were each strong predictors of T8 smoking behavior. Subjects who had left school were smoking at more than twice the rate of subjects still in high school (grade 12) at T8. We discuss implications of the results. (*Am J Public Health* 1989; 79:1371–1376.)

Introduction

The social influences approach to smoking prevention has proven efficacious in a large number of studies.[1,2] The approach concentrates on making students aware of the social pressures to smoke—from peers, parents and other adults, and the mass media—and on teaching them behavioral skills with which to resist such pressures. Most studies have reported about 50 percent reduced levels of smoking onset by students exposed to social influences programs when compared to control students, at least in the short term. Ultimately, however, the effectiveness of the tested programs, and of the social influences approach generally, must be assessed from long-term results. We must be able to reduce the prevalence of smoking in high school and beyond.[3]

Long-term Effects

To date, only a few investigators have reported results of social influence interventions into high school or beyond two years after the intervention (Appendix A).[4–10] Reported effects become smaller at longer follow-ups, until they disappear altogether at six-year follow-up.[10] Results in all cases are difficult to interpret because of alternative methodological explanations.[1,2] These methodological problems were overcome in "third generation" studies,[1,2] of which the Waterloo study was one. The data reported here are the first long-term follow-up data to be reported from a "third generation" study.

The Waterloo Study

The first large-scale randomized trial of the social influences approach to smoking prevention was initiated in Waterloo, Canada in 1979. At that time, 22 schools wit[illegible] de 6 students were randomly assigned to receive or not receive a social influences curriculum. Grade 6 students in the 11 program schools received a 6-session curriculum from research staff. Two maintainence sessions were provided at the end of grade 6, two booster sessions in grade 7, and one booster session in grade 8. Appendix B provides a summary of curriculum content, and further details are provided in previous publications.[11–13]

Students in both program and control schools were assessed by questionnaire at pretest (T1), immediately after the core program (T2), at the end of grade 6 (T3), at the beginning and end of grade 7 (T4 and T5), and at the end of grade 8 (T6). Potential underreporting of self-reported smoking behavior was minimized by assurances of confidentiality, and collection of a sample of saliva as a modified "bogus pipeline."[14] Results indicated that although the program did not reduce levels of regular smoking or significantly increase the probability of remaining a nonsmoker, it was successful at preventing the onset of experimental smoking up to the end of grade 8.[12,13] Results were especially encouraging for high-risk students—those at risk to become smokers because they had already tried smoking at grade 6 or because their parents, siblings, and friends were smokers.[11]

Methods

Subjects

Participants in the first Waterloo smoking prevention trial were reassessed five and six years after the beginning of the study, in grades 11 (T7) and 12 (T8). Extensive tracking procedures resulted in the location of a large percentage of the original subjects. For the 11th grade (T7) assessment, 79 percent (223) of the original 281 subjects from one school board were located and 68 percent were tested. However, failure to obtain cooperation from one school board in time resulted in a failure to assess many subjects in grade 11, and only 40 percent (166) of the 412 subjects from the second school board were located, and 35 percent (143) tested. Overall 56 percent (389) of the subjects were located and 48 percent (335) tested.

The following year we obtained increased cooperation from the second school board. This enabled us to locate 623 (90 percent) of all study students and to obtain questionnaire data from 532 (77 percent) of them and telephone information from an additional 28 (4 percent). Therefore, all outcome results reported below are limited to data from T8 when we obtained responses from most subjects.

Measures

Measures used at T8 were essentially the same as those previously used in grades 6 through 8. In addition to self-reported smoking behavior, students and school leavers were asked for demographic information; reports of smoking habits of parents, siblings, and friends; and information on a

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set of mediating variables including knowledge and beliefs regarding smoking, environmental change (from grade school to high school), other substance use, and academic aspirations. Students were also asked for reports of smoking habits of teachers, and information on school environment (including smoking regulations and enforcement).

Smoking behavior categories—On the basis of self-reported smoking behavior, subjects were classified as being in one of five behavioral categories at pretest and at the 12th grade follow-up:

- Never smoker (never smoked even one puff of cigarette);
- Tried once (has smoked, but only once);
- Quitter (has smoked more than once, but has "quit for good");
- Experimenter (currently smokes, but less than once a week);
- Regular (currently smokes at least once a week or more).

Some analyses reported below compare regular smokers against all others. For some analyses, we also classified subjects as to whether or not they were currently smoking at all.

Pretest social environment risk—Risk levels were based on pretest responses to questions about the smoking habits of parents, siblings, and friends. Levels were defined as follows:

- Low risk (no smoking parents, siblings, or friends);
- Intermediate risk (smokers in only one of the three social model groups);
- High risk (smokers in two or all three social model groups).

Both the behavior smoking category and the social environment classifications depend on multiple questionnaire responses. If needed items were missing, the subjects were unclassifiable and excluded from the analyses. Over 93.5 percent had smoking behavioral categories for both pretest and 12th grade and 91 percent had complete data for all variables used in the analysis.

Procedure

Most subjects were still in school, and in the high schools they expected to be in when they last participated in the study. However, a significant number had changed schools or dropped out. Extensive tracking procedures were initiated to find and assess as many of these students as possible. Those students who had left school or had moved to another area were surveyed by mail (see reference 15 for details).

Those students located in schools in the vicinity of the University of Waterloo were surveyed in classrooms. Participation in measurement was by full informed consent by both the students and their parents. Confidential ID numbers enabled us to match student responses across waves of data collection.

Results

Attrition Analyses

Measures taken at T1 were used in a logistic model[16,17] to determine whether subjects lost at T8 differed systematically from those who were retained. Effects analyses could be biased to the extent that variables related to outcome behavior also relate to the process of attrition. There was no significant relation between treatment condition and attrition. Neither was there a relation between pretest social environment risk and attrition. However, there was a relation between pretest smoking behavioral category and attrition, with greater attrition of pretest triers compared to never smokers (odds ratio = 1.84, 95% CI = 1.04, 3.28). Age was also related to attrition. The study retained more subjects who were ages 9–11 at pretest compared to those who were age 12 or older (OR = 2.53, 95% CI = 1.45, 4.39). (These older subjects were more likely to have left school and were thus more difficult to track.) As both students smoking at pretest and older students were more likely to be smoking at the time of the follow-up, these differences tend to bias downward observed smoking rates across all conditions.

Smoking Levels

Approximately 33 percent of all subjects at T8 were regular smokers (95% CI = 27.95, 38.72) and another 10 percent were experimental smokers (95% CI = 6.63, 13.49). By this time only 15 percent had never tried a cigarette (95% CI = 11.07, 19.26).

Students and school leavers—Students and school leavers reported very different patterns of smoking behavior. Approximately 68 percent of school leavers were regular smokers compared to 28 percent of students still in school at grade 12 (OR = 5.54, 95% CI = 3.14, 9.79). Nearly all of the school leavers (98 percent) reported having tried smoking compared to only 84 percent of the students (OR = 12.50, 95% CI = 1.71, 91.8). Figure 1 presents smoking behavioral categories for students in grade 12 and for school leavers.

Measures taken at T1 were used in a logistic model to predict school leaving six years later at T8. There were no significant differences between students and school leavers by original school board, treatment condition, or pretest social environment risk. There were, however, differences by age and by pretest behavioral smoking category. Most of the students were 11 years old at the time of the intervention, but a few were as young as 9 years or as old as 14. Students who were age 12 or older at the time of the pretest were more likely to have left school than those who had been ages 9–11 (OR = 5.05, 95% CI = 2.53, 10.08). Those who had tried smoking once at the time of the pretest were more likely to leave school than those who had never smoked (OR = 2.95, 95% CI = 1.38, 6.29), as were those who had quit (OR = 3.21, 95% CI = 1.30, 7.91) and those who had been regular smokers at the pretest (OR = 7.49, 95% CI = 2.15, 26.09).

However, age and pretest smoking rate differences cannot account for all differences between smoking rates of students and school leavers at the T8 follow-up. When age,

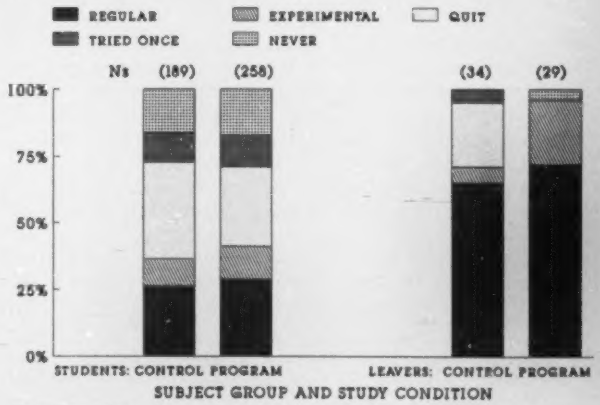

FIGURE 1—Percentage of Grade 12 Students and School Leavers in Smoking Behavior Categories

TABLE 1—Logit Model Summary Table for 12th Grade (T8) Smoking

T1 Predictor of Smoking at T8	Odds Ratio	(95% CI)
Intercept	0.26	(0.15, 0.46)
Smoking Category		
Tried vs Never	1.97	(1.27, 3.06)
Quit vs Never	2.70	(1.46, 4.99)
Experiment vs Never	4.53	(2.19, 9.37)
Regular vs Never	14.35	(3.09, 66.68)
Social Environment Risk		
Intermediate vs Low	1.71	(0.97, 3.02)
High vs Low	2.23	(1.22, 4.07)
Original School Board		
Waterloo vs Oxford	0.69	(0.47, 1.01)
Treatment Condition		
Treatment vs Control	1.22	(0.83, 1.80)

TABLE 2—Logit Model Summary Table for 12th Grade (T8) Regular Smoking

T1 Predictor of Regular Smoking	Odds Ratio	(95% CI)
Intercept		
Smoking Category		
Tried vs Never	1.67	(1.0, 2.70)
Quit vs Never	3.69	(1.96, 6.93)
Experiment vs Never	4.86	(2.3, 9.92)
Regular vs Never	7.09	(2.2, 22.38)
Social Environment Risk		
Intermediate vs Low	1.12	(0.61, 2.06)
High vs Low	1.75	(0.93, 3.29)
Original School Board		
Waterloo vs Oxford	1.56	(1.03, 2.35)
Treatment Condition		
Treatment vs Control	1.24	(0.83, 1.86)

pretest behavioral smoking category, social environment risk, original school board and treatment condition are included with school leaving in a logistic model to predict regular smoking at T8, there is still more regular smoking by school leavers than by students (OR = 2.18, 95% CI = 1.58, 3.01).

Program Effects

Overall program effects also were assessed using a multiple logistic model. The dependent variable is smoking at T8. The primary predictor is treatment condition. The model includes smoking behavioral category at the 6th grade pretest (T1) and pretest social environment risk as additional predictors. These were previously shown to be effective predictors of smoking at the end of 8th grade (T6).[11,13] Original school board is included as a blocking factor for possible differences between the two populations. Table 1 summarizes the results of this analysis. Interactions of treatment condition with each of these two variables then were added to assess possible differential program effects for students at differing degrees of risk. These interactions were not significant.

By T8 there is no longer a significant overall effect for the treatment program. Both pretest social environment risk ($p <$ .05) and pretest smoking behavior ($p < .0001$) predict whether students are smoking more than six years later. The odds ratio indicate that those at low social environment risk in grade 6 are least likely to be smoking six years later. There is no significant difference in smoking rates between the two original school districts. Since the interactions are insignificant, there do not appear to be any differential effects of the treatment program for students at different initial levels of risk for smoking. The overall likelihood-ratio test (*Chi-square* = 30.86, $df = 38$, $p > .75$) indicates that the terms in the model are adequate to account for the observed patterns of responses.

A parallel analysis was done using regular smoking as the outcome measure. Table 2 summarizes these results. A similar pattern of effects is observed for regular smoking (at least once a week) as for smoking at all. There are no overall program effects nor differential program effects for students at differing initial levels of risk. Pretest smoking behavior category ($p < .0001$) predicts whether a student is a regular smoker at T8. Pretest social environment risk is a less strong predictor ($.05 < p < .10$) but the odds ratio are in the same direction for regular smoking as they were for smoking. The likelihood-ratio test (*Chi-square* = 43.42, $df = 38$, $p > .25$) indicates that the terms in the model are adequate to account for the observed patterns of responses.

Thus, six years after the beginning of the intervention, there were no overall differences between the program and control groups when all students are considered together. Figure 2 displays the proportion of subjects smoking regularly and experimentally at each wave by experimental condition. It demonstrates decay of the significant difference between conditions in level of experimental smoking that was observed at grades 7 and 8.[13]

Considering pretest smoking behavior and social risk categories,[11] Figures 3 and 4 show how pretest risk is a strong predictor of subsequent smoking level. The probability of regular smoking in high school increases as grade 6 risk increases; the probability of experimental smoking in high school is lowest for the students at lowest risk in grade 6, and the probability of never smoking by grade 12 decreases as grade 6 risk increases. At grades 7 and 8, we observed significant program effects for the high- and middle-risk students—we did not observe effects for the low-risk students because only a few of them had tried smoking by grades 7 and 8. We observed no significant differential program effects by 12th grade for any risk group.

Discussion

Observed effects of the Waterloo program on the onset

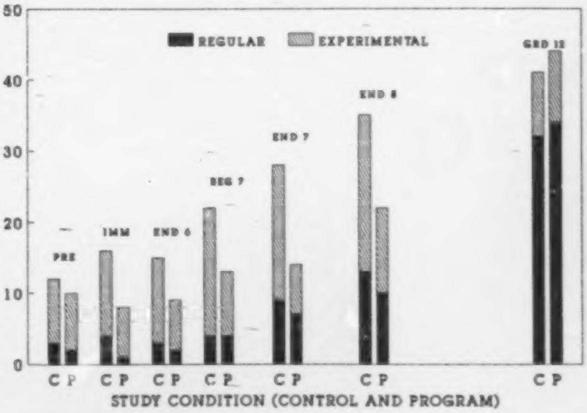

FIGURE 2—Proportion of Subjects Smoking Regularly and Experimentally at Each Wave of the Study

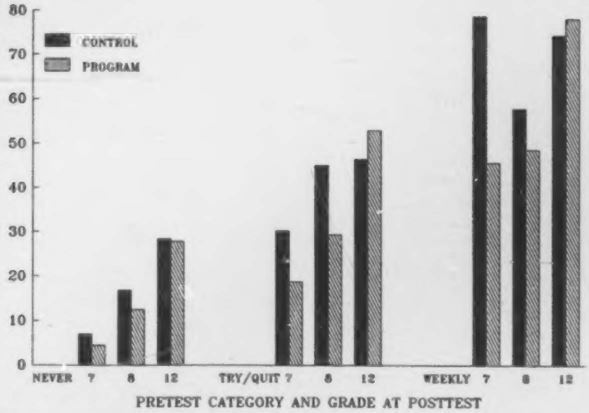

FIGURE 3—Percentage of T8 Subjects Smoking by Pretest Smoking Category and Experimental Condition

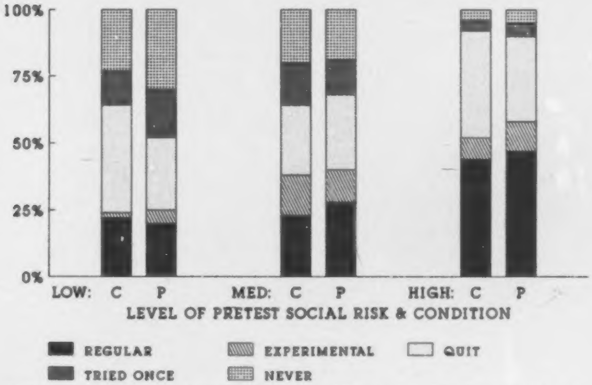

FIGURE 4—Percentage of T8 Subjects in Each Behavioral Category by Pretest Social Risk Level and Experimental Condition

of experimental smoking in grades 7 and 8 were not maintained through grade 12. Expected effects on grade 12 smoking (regular or experimental) did not occur. This finding is consistent with that of the only other study of long-term effects of adolescent smoking prevention programs.[9,10]

The lack of significant preventive effects by grade 12 raises the question of the value of the social influences approach for smoking prevention. In retrospective non-intervention studies, delayed onset is associated with improved prognosis for quitting and lower incidence of total morbidity and mortality.[18,19] Prevention program-induced delays in onset may reduce total lifetime exposure and may be an important outcome.[10] This is particularly true if delayed onset increases the probability of earlier quitting. However, the extent to which an intervention-induced delay in onset is associated with earlier or more successful quitting, or even with decreased exposure to toxins, is not established. That would require following the subjects of this study, and others like it, into middle adulthood. We might expect to find earlier quitting by subjects who are currently smokers but who delayed onset until grade 9 or 10, partly because of the delayed onset and partly because of reduced peer influences.

The value of the social influences approach to preventing the onset of regular smoking by the end of high school needs further study. Results from both the Minnesota and Waterloo studies suggest that program effects obtained in junior high school decay gradually during the following years to totally disappear by grade 12. Such a pattern suggests that booster sessions might be necessary. However, the Waterloo results suggests that boosters in grade 8 are insufficient. These results, together with findings of increased risk at the transition from one level of school to another[20] (or from school to college or working), and the importance of school policy variables[21] suggest the need for boosters in high school. Results of reported high school interventions[6,22,23] suggest that social influence curricula can be effective with high school students, although effects were small in all cases. Booster sessions were also recommended by an expert advisory panel convened recently by the National Cancer Institute.[24]

The apparent lack of effects of social influence programs on smoking prevention by grade 12 should not be over-interpreted. First, as noted above, boosters in early high school years may help to maintain early substantial effects. Second, there is a much better understanding today than 10 years ago of the essential components of effective prevention programs.[24] These improvements may well mean that current versions of social influence programs may produce more durable effects. Third, the broader social norms are now more supportive of nonsmoking, so that students in control schools may, in fact, have been exposed to many of the elements of the social influence approach during the last five years.

One alarming result of this study is the large difference in smoking behaviors between students and school leavers. These differences cannot be explained by differences in age or by pretest differences in smoking risk or experience, and they also replicate results from the Minnesota group.[25] Indeed, they may be underestimated in both the Minnesota and this study because school leavers were followed up less successfully than subjects still in school. The high rates of smoking by early school leavers or dropouts warrant special attention by future research. Prevention programs for youth who will leave or drop out of school will need to go beyond the school setting. Early cessation programs also need to be developed for this group, and these will require components designed to motivate young adults to consider quitting, something which most current smoking cessation programs do not include.

Additional studies of the long-term effects of social influence prevention interventions are necessary. We cannot draw strong inferences from only the two long-term follow-up studies conducted to date. The short-term effects of the social influences approach appeared very promising, but interpretation of their long-term value must await further long-term follow-up studies.

APPENDIX A

Results and Characteristics of Long-term or High School Follow-up Studies on Smoking Prevention

Authors/ Date/Reference	Length of Follow-up (Years)	School Grade of Program	Program Effect (Yes or No)	Methodological Problems (See below)
Evans, *et al*, 1981[4]	3	7	Y	a,b
Telch, *et al*, 1982[5]	2	7	Y	c,d
Johnson, *et al*, 1986[6]	1	10	Y	a,e
Hansen, *et al*, 1988[7]	3	7	Y	a,e
Luepker, *et al*, 1983[8]	2	7	Y	f
Murray, *et al*, 1988[9]	4–5	7	Y	f
Murray, *et al*, in press[10]	5–6	7	N	f

Methodological problems:
a) Serious attrition.
b) Reliance on successive cross-sectional analyses of differenct subjects.
c) Pretest differences.
d) Unable to follow same subjects over time.
e) Marginal significance.
f) Pretest differences in direction of expected effects.

APPENDIX B

Summary of Content of the First Waterloo Smoking Prevention Program

Session	Components	Purpose	Methods
Core program (Six one-hour sessions)	Informational	Provide factual basis for the rest of the program.	Film Classroom discussion Poster-making Videotapes Categorization
	Skills Development	Develop and practice skills to resist social influences to smoke.	Videotapes Modeling Role playing Poster-making
	Decision-making	Integrate other components and express intentions.	Decision sheet Public commitment to class
Maintenance (same grade)		Reestablish contact Review	Poster-making Classroom discussion "Talking" videotape Categorization
Booster sessions (grades 7 & 8)		Reestablish contact Review Update information and skills	Classroom discussion Small group work Decision sheet Role playing

ACKNOWLEDGMENTS

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Portions of these data were previously presented at the 6th World Conference on Smoking and Health, Tokyo, Japan, October 1987.

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RWJ Foundation Extends Grants Program for Native American Health Projects

The Robert Wood Johnson ~~Foundation~~ recently awarded nearly $2 million to fund 13 community health care projects run by and for American Indians and Alaska Natives, and in a further commitment has allocated additional funds to extend the program through 1990.

Believed to represent the first commitment by a major foundation to work directly with tribal governments, the 1989 grants program will target projects designed to prevent alcohol and drug abuse, control diabetes, reduce domestic violence, and improve maternal and infant health among tribal populations in eight states (AZ, ID, MN, MT, NM, OK, SD, and WI). The tribes plan to approach their health problems with imagination and creativity, using traditional Indian methods of care as well as modern medical technology, according to the foundation.

When the call for proposals was issued last fall for the program, "Improving the Health of Native Americans," the foundation received applications from Indian-related groups in 31 states. The high quality and magnitude of that response prompted the foundation to commit additional funds of up to $2 million to continue the program in 1990.

Tribes and related community organizations interested in applying for funds under the program's second phase should contact Timothy L. Taylor, PhD, Program Director, Improving the Health of Native Americans, College of Public Health, University of Oklahoma Health Sciences Center, PO Box 26901, Oklahoma City, OK 73190. Tel: (405) 271-3221. Dr. Taylor is also a member of the Kiowa Tribe.

Two workshops for potential applicants will be held in November in Nashville, TN and Denver, CO, to answer questions about the program and the application process. For more information about the workshops, contact Dr. Taylor at the address above.

Grants under the program's second phase will be announced in August 1990; proposals must be received by February 1, 1990 to be considered for funding.

The RWJ Foundation, based in Princeton, NJ, is the nation's largest health care philanthropy, established in 1972. Since then, it has awarded more than $996 million in grants to improve health care in the United States.

Elevated Blood Levels of Carcinogens in Passive Smokers

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AND STEVEN R. TANNENBAUM, PHD

Abstract: The hypothesis that involuntary exposure to tobacco smoke—passive smoking—results in greater risk of cancer was assessed by measuring the levels of two known carcinogens in the blood of 57 nonsmokers with varying degrees of involuntary exposure, including six heavily exposed bartenders. The concentrations of hemoglobin adducts of 4-aminobiphenyl, a bladder carcinogen, were significantly higher in subjects with confirmed involuntary exposure (plasma cotinine concentrations between 2 and 23 ng/ml) compared with subjects with undetectable levels of cotinine. Similarly, adducts of 3-aminobiphenyl were significantly elevated in subjects with confirmed exposure. The odds of 3-aminobiphenyl adduct levels exceeding 2 pg/g of hemoglobin were 6:7 among the confirmed exposed, compared with the odds of 2:42 among subjects with undetectable cotinine (odds ratio = 18; 95 percent confidence interval = 3.3, 94). The validity of the assay was demonstrated by showing striking declines in adduct levels among quitting smokers. (*Am J Public Health* 1989; 79:1381–1384.)

Introduction

Involuntary exposure to tobacco smoke, or "passive smoking," has been found weakly associated with lung cancer in several epidemiologic studies, but several others have found no association.[1–3] Critics argue this evidence is flawed.[4,5] The International Agency for Research on Cancer (IARC) states that: "The observations on nonsmokers that have been made so far are compatible with either an increased risk from 'passive' smoking or an absence of risk. Knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed during 'passive' smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens . . . leads to the conclusion that passive smoking gives rise to some risk of cancer."[3] Mantel[6] concludes that the relative risk of passive smoking is too low to be detected by epidemiologic studies.

If Mantel is right, assessment of the risk of passive smoking depends on measurement of the extent of absorption of carcinogens from tobacco smoke. No evidence has been reported to date of increased blood levels of a known carcinogen in passive smokers. We collected blood samples from nonsmokers with various degrees of passive exposure, classified them by plasma cotinine detectability, and measured levels of 4-aminobiphenyl and 3-aminobiphenyl covalently bound to hemoglobin. The adducts of these aromatic amines with hemoglobin (4ABP-Hb; 3ABP-Hb) are formed over the 120-day lifespan of the erythrocyte and therefore may serve as dosimeters of average exposure over the previous four months.[7] 4ABP is a known human carcinogen[8] and 3ABP, although not proven to be carcinogenic in humans, is a marker of 4ABP formation by pyrolysis[9] and has been shown carcinogenic in rats.[10]

Methods

Subjects

Nonsmokers were recruited by a variety of methods to cover a range of passive exposures. To represent chronic passive exposure, six heavily exposed nonsmoking bartenders were recruited by a telephone survey of local bars. To represent more normal but quantitatively uncertain levels of passive smoking, 21 nonsmokers who reported exposure to at least one pack of cigarettes per day were recruited by asking colleagues to survey their friends. To represent lack of exposure, 14 nonsmokers who reported negligible passive exposure in the past two months were recruited from our research staffs. Added to this was a group of blood donors in Italy who served as nonsmoker controls for another study.[11] The Italians were not selected according to their degree of passive exposure and therefore were presumed to be more representative of a cross-section of nonsmokers.

Exposure

Local volunteers, after completing a consent form, donated 10–20cc of blood, which was drawn into a vacuum tube containing EDTA (ethylenedinitrilotetraacetate) as anticoagulant and immediately placed in a cooler. Using a short standard questionnaire, subjects were interviewed about their exposure to tobacco smoke during the past two months. Questions included numbers of smokers in the work and home environment, and estimated numbers of packs of cigarettes per day smoked in their presence. Information on involuntary exposure to smoke was not available from of the Italian blood donors.

Plasma cotinine was measured by the laboratory of N.J. Haley of the American Health Foundation, Valhalla, NY. Four subjects were excluded for having cotinine levels that were suspicious of active smoking[12]: One local volunteer who claimed only passive exposure had 240 ng/ml of cotinine, and three Italians who had reported being nonsmokers had 61, 97, and 101 ng/ml of cotinine in their plasmas. The remaining subjects were divided into two groups: 1) undetectable cotinine, and 2) detectable cotinine. (The limit of detection of the cotinine assay was 2 ng/ml.) These two groups were then subdivided according to the level of exposure they reported in their interviews, as follows.

Group 1a (n = 14): Research workers; no cotinine; reported little or no passive exposure.
Group 1b (n = 15): Italian blood donors; no cotinine; no interview about passive exposure.
Group 1c (n = 15): Local volunteers, no cotinine; reported passive exposure to at least one pack of cigarettes per day.

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Group 2a (n = 7): Local volunteers; measurable cotinine; reported passive exposure to at least one pack of cigarettes per day, plus one Italian blood donor with measurable cotinine.

Group 2b (n = 6): Local bartenders; measurable cotinine; reported passive exposure to five or more packs of cigarettes per day.

Group 1b was ranked between Groups 1a and 1c because it presumably comprised a mixture of people who would have been assigned to Groups 1a or 1c if they had been interviewed.

Adduct Assay

The methods of sample preparation and analysis for determining the concentration of 4ABP adducts with hemoglobin have been described in detail.[13,14] In brief, hemoglobin was isolated from red blood cells and purified by dialysis. The dialysate was spiked with a nanogram of an internal standard (4′-fluoro-4-aminobiphenyl) and then hydrolysed with dilute base to release free 4ABP and 3ABP from the adducts. The amines were extracted into purified hexane and then derivatized to form the corresponding pentafluoropropionamides. These were quantified using capillary gas chromatography with detection by negative-ion chemical ionization mass spectrometry. The average limit of detection of the assay for 3ABP was 1.0 pg/g of Hb, calculated as the quantity of adduct necessary to produce a peak intensity three times the root mean square of the individual chromatogram's baseline noise level.

The precision of the assay was assessed by splitting two 100 ml blood samples into 8 and 7 aliquots.[14] The coefficient of variation of the estimates of 4ABP-Hb concentration, expressed in pg/g of Hb, was 10 percent for each sample. In addition, from 15 people with varying levels of 4ABP-Hb, duplicate blood samples were collected 15 or 48 hours apart. The coefficient of variation in 4ABP-Hb adduct concentration among duplicates was 9 percent, entirely attributable to variation in the assay technique. This suggested day-to-day biologic variation in adduct levels was negligible.

Validation Studies

To demonstrate the validity of the level of the hemoglobin adducts as dosimeters of human exposure, we recruited seven quitting smokers and measured adduct levels in blood samples drawn on the day they entered the stop-smoking program, and two months or more later. Serum cotinine was used to confirm that all seven had successfully quit. Further validation data were found in the results of a cross-sectional study conducted in Italy in which the dose-response relationship between intensity of smoking and adduct level was examined.[11]

Data Analysis

Since the distributions of adduct levels among passive smokers and unexposed nonsmokers were found to be skewed, nonparametric tests were used. The median two-sample test was the preferred basis for inference because it is relatively insensitive to outliers.[15] The Wilcoxon two-sample test was also used.[15] One-sided p-values were used because the hypotheses were directional.

Subjects with undetectable levels of 3ABP-Hb were handled in two ways. First, the subjects were assigned the highest level of 3ABP that could have gone undetected, given the noise level of each chromatogram. Second, the subjects were omitted from the analysis.

Results

Validity

We found striking reductions in adduct levels among quitting smokers. The mean level of 4ABP-Hb fell 75 percent from 130.4 pg/g Hb (s.e. = 35.1 pg/g Hb) at baseline to 33.3 pg/g Hb (s.e. = 7.3 pg/g Hb) after 2 months. The mean level of 3ABP-Hb fell at least 80 percent from 16.0 pg/g Hb (s.e. = 7.2 pg/g Hb) to a baseline of approximately 1.7 pg/g Hb (s.e. = 1.3). Only three of the seven subjects contributed to the estimate of the average final level of 3ABP-Hb because concentrations in the other four subjects were below the limits of detection of the assay.

The cross-sectional study of Italian smokers[11] showed dose-response relations existed between number of cigarettes smoked per day and levels of both 4ABP and 3ABP.*

Passive Smokers

The characteristics of each subgroup and the median and mean levels of 4ABP-Hb and 3ABP-Hb are shown in Table 1. The individual levels of 4ABP-Hb and 3ABP-Hb are plotted in Figures 1 and 2.

Levels of 4ABP-Hb were marginally higher in Group 2 than in Group 1 (Figure 1). The difference was of borderline statistical significance: the median two-sample test statistic was 1.64 (p = 0.05). The Wilcoxon two-sample test statistic was only 1.08 (p = 0.14) due to several atypically high values in Group 1. The highest value (152 pg/g) was from a woman who claimed passive exposure to 10 cigarettes per day but had no detectable cotinine in her blood. The next two highest values were from women in Group 1a. One woman with 89 pg/g confirmed on reinterview that she had had absolutely no

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*Data available on request to author.

TABLE 1—Levels of 4-Aminobiphenyl and 3-Aminobiphenyl Adducts with Hemoglobin in Groups of Nonsmokers with and without Detectable Serum Cotinine, According to Level of Reported Passive Smoking

						4-Aminobiphenyl*			3-Aminobiphenyl*†		
Group	Detectable Cotinine	Reported Passive Smoking	Number of Subjects	Mean Age	Percent Male	Median	Mean	(S.E.)	Median	Mean	(S.E.)
1a	No	None	14	37	43	40	50	24	1.0	1.3	1.5
1b	No	—	15	39	100	37	42	15	1.0	1.2	0.6
1c	No	Yes, Low	15	28	26	38	46	31	1.2	1.2	0.7
2a	Yes	Yes, Low	7	32	86	42	45	14	0.7	1.5	1.3
2b	Yes	Yes, High	6	31	83	48	54	21	2.6	2.4	1.1

*Picograms of amine per gram of hemoglobin.
†Minimum detectable levels were used for subjects with undetected levels.

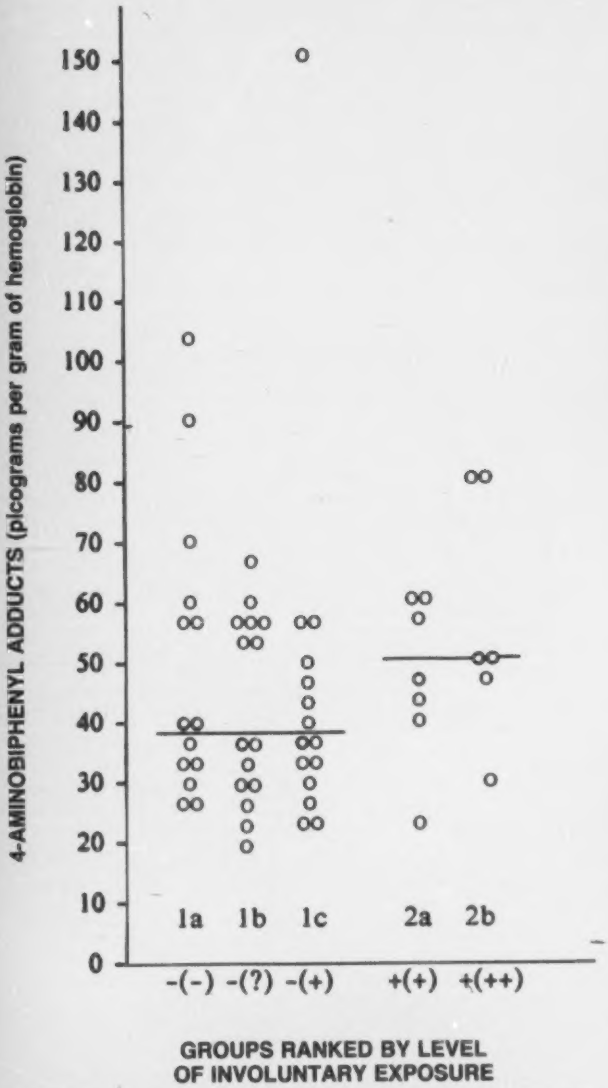

FIGURE 1—Levels of 4-Aminobiphenyl Adducts with Hemoglobin in Groups of Nonsmokers without (1a, 1b, 1c) and with (2a, 2b) Detectable Serum Cotinine, Indicated by − and + Signs.
Symbols in parentheses refer to self-reported level of passive exposure to tobacco smoke: (−) none; (?) unknown; (+) low; (++) high. Horizontal lines indicate locations of group medians.

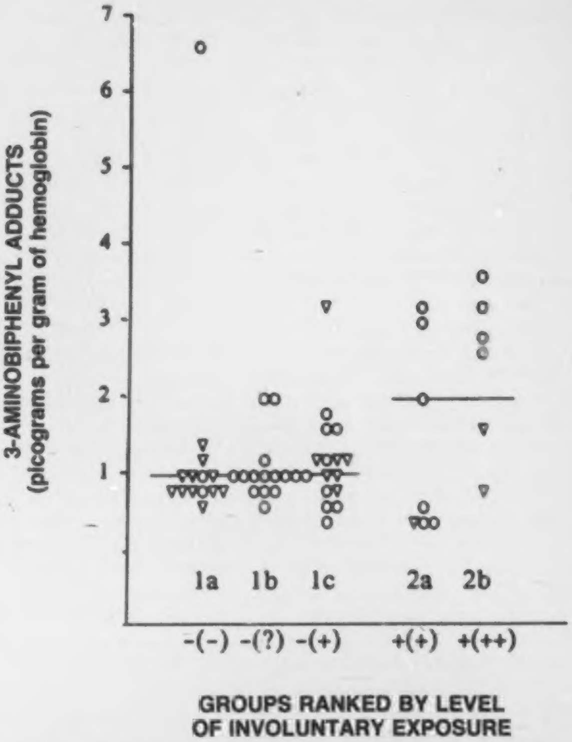

FIGURE 2—Levels of 3-Aminobiphenyl Adducts with Hemoglobin in Groups of Nonsmokers without (1a, 1b, 1c) and with (2a, 2b) Detectable Serum Cotinine, Indicated by − and + Signs.
Symbols in parentheses refer to self-reported level of passive exposure to tobacco smoke: (−) none; (?) unknown; (+) low; (++) high. Horizontal lines indicate locations of group medians. Triangles represent undetectable levels of adduct and are plotted at the highest level the subject could have had without detection.

passive exposure. Another, with 103 pg/g, reported only occasional mild exposure for short periods. Increasing the size of the control group by adding the seven exsmokers from the validation study resulted in a mean two-sample test statistic of 1.54 ($p = 0.06$) and a Wilcoxon two-sample test statistic of 1.41 ($p = 0.08$).

A clearer difference in the hypothesized direction was found for 3ABP-Hb levels (Figure 2). Group 2 had significantly higher levels than Group 1. The median two-sample test statistic was 1.92 ($p = 0.027$) and the Wilcoxon two-sample test statistic was 1.22 ($p = 0.11$), using the lowest detectable level for subjects with undetectable adduct levels. When undetectable levels were omitted, the median two-sample test statistic was 2.10 ($p = 0.017$) and the Wilcoxon two-sample test statistic was 1.65 ($p = 0.05$). The odds of a subject with detectable cotinine having a level of 3ABP-Hb greater than 2 pg/g of Hb were 6:7, compared with 2:42 among subjects with undetectable cotinine. The odds ratio was 18 with a 95 percent confidence interval from 3.3 to 94.

Discussion

Our observations corroborate the hypothesis that involuntary exposure to tobacco smoke results in higher levels of carcinogens in the blood. Subjects with plasma cotinine concentrations between 2 and 23 ng/ml had significantly higher concentrations of 4ABP-Hb and 3ABP-Hb than subjects with undetectable levels of cotinine.

The association with 4ABP-Hb was of only borderline statistical significance. However, it is less likely to be due to chance than the p-values indicate because the significant association with 3ABP-Hb provides additional evidence against the null hypothesis. It is well established that 3ABP forms in the same pyrolysis reactions as 4ABP.[9] Consistent with this, our validation study showed that 4ABP-Hb and 3ABP-Hb both declined among quitting smokers. Therefore, 3ABP-Hb is in part a marker of 4ABP exposure.

Nevertheless, the magnitude of the elevation in adduct levels was small relative to normal background levels. Nontobacco sources of 4ABP, and perhaps 3ABP, appear to exist. This was underscored by the fact that one woman with absolutely no passive exposure to tobacco smoke had levels of 4ABP-Hb higher than all the heavily exposed bartenders. Her level of 3ABP-Hb was undetectable, but another woman with minimal passive exposure had equally high 4ABP-Hb and the highest level of 3ABP-Hb in the study.

Since it is well established that 4ABP causes bladder cancer in humans,[8] our data suggest that risk of bladder cancer among heavily exposed passive smokers is slightly elevated. However, the increase in risk may be too small to detect in an epidemiologic study. A relative risk of 1.2 is regarded by many as the lower limit of detection in nonexperimental epidemiology. It is hard to detect a relative risk of 1.1 not just because of sampling variation but because of uncontrolled confounding. A potential confounding factor weakly associated with disease (relative risk = 1.7) could produce a spurious relative risk of 1.1 for passive smoking if passive smokers were twice as likely as unexposed nonsmokers to be exposed to the confounder.[16] A relative risk of 1.1 corresponds to a 10 percent excess in risk. For passive smoking to cause at least 10 percent excess in risk of bladder cancer, when active smoking causes an excess of only 100 percent (i.e., relative risk = 2), the excess concentration of activated carcinogens in the tissues of passive smokers should be approximately 10 percent of the excess in active smokers (assuming a linear relation between *in situ* dose and risk). In our data, the excess of adduct attributable to passive smoking appeared to be lower than this. Whereas the median level of 4ABP-Hb adducts in active smokers was found to be 200 percent to 400 percent in excess of the median level in unexposed nonsmokers,[10,13] the median level among passive smokers was only 10 percent to 20 percent in excess (i.e., the excess in passive smokers was 2.5 percent to 10 percent of the excess in active smokers).

Since our data are few, especially among confirmed passive smokers, and the assumption of linearity between adduct levels and risk is hypothetical, firm conclusions cannot be drawn. However, when compared with data that indicate cotinine levels in passive smokers are generally 1 percent, and at most 2–10 percent, the levels in active smokers,[3,17] our data seem more compatible with an undetectable rather than a detectable relative risk of bladder cancer due to passive smoking. One conclusion that does seem warranted, however, is that 4ABP does not conform to Remmer's hypothesis.[18] Remmer suggested that a larger proportion of inhaled carcinogens are metabolically activated in passive smokers, as compared to active smokers, due to the induction of detoxifying enzymes by substances in mainstream smoke that are not found in sidestream smoke. This does not appear to hold for 4ABP, assuming that the adduct with hemoglobin is a marker of the levels of activated 4ABP. There is evidence to support such an assumption.[13,19]

It is important to emphasize that "undetectable" does not mean "nonexistent." Since involuntary exposure to tobacco smoke is very common, even an undetectable relative risk such as 1.1 would be responsible for a great deal of avoidable cancer. The purpose of distinguishing between detectable and undetectable relative risks is for planning research to quantify the risk of passive smoking. If studies like ours show that post-activation levels of other tobacco carcinogens are much lower in passive smokers than active smokers, it will add weight to the view that quantification of the risk of passive smoking is beyond the capability of questionnaire-based epidemiologic studies, except when the association between active smoking and disease is very strong (e.g., relative risk of 10 for lung cancer). Risk assessment may instead depend on biochemical studies of carcinogen metabolites.

In conclusion, our results corroborate both the statement by the IARC that "passive smoking gives rise to some risk of cancer" and critics' contention that relative risks due to passive smoking may be undetectable.

ACKNOWLEDGMENTS

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Delayed Blood Regeneration in Lead Exposure: An Effect on Reserve Capacity

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Abstract: Twenty-five lead-exposed Danish battery production workers and 25-age-matched controls were examined to evaluate subclinical effects on blood formation. Blood lead levels averaged 2.14 μmol/L and 0.35 μmol/L in the two groups; the lead workers also showed high levels of erythrocyte protoporphyrin, as compared to the controls. Otherwise, the hematological parameters indicated an appropriate iron status and no other deviations.

From all subjects, 0.45 L of blood was bled as part of a normal blood donation. Five and 11 days later, reticulocyte counts were significantly higher in the control group than in the lead-exposed workers. On day 15, the lead workers showed a significant delay in blood regeneration, as evidenced by lower hemoglobin concentration, and erythrocyte and reticulocyte counts.

The lead exposure in the present study was within legal limits, and lead-induced anemia would be expected only at much higher exposure levels. Thus, despite the normal hematological findings in the initial examination, the lead exposure caused a decreased reserve capacity for blood formation, and this effect became evident only after the blood loss. (*Am J Public Health* 1989, 79:1385–1388.)

Introduction

Although lead is one of the most extensively studied pollutants, the health significance of early hematological changes at slightly increased exposures remains unclear. Thus, heavy lead exposures are associated with low hemoglobin or anemia.[1,2] However, when blood-lead levels do not exceed 2.9 μmol/L, hemoglobin concentrations in male lead workers are usually entirely within normal ranges.[1–3] Despite the apparent absence of hematological abnormalities, such lead exposures reduce the lifespan of the erythrocytes and inhibit the heme biosynthesis.[4,5] These biochemical effects in a lead-exposed worker could place an increased strain on the bone marrow to keep the red-cell values within normal ranges.

Therefore, increased demand on the formation of blood following blood loss could result in a delayed blood regeneration in individuals exposed to lead. Only one experimental study has examined this question: dogs exposed to lead in the feed showed no delay in blood regeneration after a controlled bleeding.[6] However, lead exposure caused an unanticipated increase in the pre-hemorrhage hemoglobin as compared to controls in this study, and dogs may well differ from humans in their reactions to lead.

We have examined the blood regeneration following phlebotomy in men with occupational lead exposure and an age-matched control group.

Methods

Sample and Research Design

Based on a review of occupational lead exposures at nearby industrial facilities, two plants were selected for study: a battery factory with high lead exposures, and a tin can factory with minimal lead exposures. The safety committees at the two facilities helped identify possible volunteers among the long-term employees. Detailed information about the study was given to each participant. At each facility, informed consent was obtained from 25 healthy men who had never been blood donors before. The median duration of occupational lead exposure at the battery plant was seven years. The comparison group at the tin can factory was age-matched (within five years) with the participants at the battery plant. The median age in the exposed and the control groups was 39 and 36 years, respectively. The median length of employment in the control group was 10 years. Information on iron intake was not included in the matching criteria.

Phlebotomy (0.45 L) was performed in cooperation with the Blood Bank at the University Hospital. Due to practical constraints, the two groups went through the procedures at two different points of time; strict quality assurance efforts were therefore applied to ensure comparability of the results. Blood lead (B-Pb), erythrocyte Zn-protoporphyrin (ZPP) and indicators of iron status were determined only in samples obtained at the time of phlebotomy. Routine hematological parameters were subsequently assessed at days 5, 11, and 15 after phlebotomy; at day 15, signs of considerable blood regeneration would be expected.[7–10]

The protocol was prepared in accordance with the Helsinki II Declaration and reported to the local ethics committee.

Blood Lead

Blood samples for lead analysis were taken in Vacutainer vials with Na-heparin (dark-blue stoppers) (Becton-Dickinson, Rutherford, NJ) under conditions minimizing the risk of sample contamination. The samples were frozen at −80°C until analysis. B-Pb analysis was carried out by electrothermal atomic absorption spectrometry.[11] We used a Perkin-Elmer model 5000 instrument with Zeeman background correction, graphite furnace HGA-500, and autosampler AS-40 (Perkin-Elmer, Norwalk, CT). All results were read against a blood-based standard curve with standard reference material SRM 955 from the US National Bureau of Standards as primary standard. The total analytical imprecision was estimated to be 8.1, 2.1 and 1.9 percent at blood lead levels of 0.24, 1.63 and 2.96 μmol/L, respectively. Participation in international external quality assurance schemes showed deviations from the overall median between −8 and +11

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percent for 14 blood samples, thus indicating a satisfactory accuracy.

Hematology

Hematological parameters were determined on fresh samples drawn in Venoject vials with Na_2EDTA (Venoject, Terumo Europe, Leuven, Belgium); special quality control procedures were used to minimize day-to-day variation.

ZPP was measured in oxidized blood on an Aviv hematofluorometer (Aviv, Lakewood, NJ).[12]

Erythrocyte count and hemoglobin concentration were determined on a Coulter Counter S using Coulter reagents (Coulter Electronics, Hialeah, FL). The instrument was whole-blood calibrated regularly and prior to each analytical series, and the calibration was controlled using the Coulter 4C Normal calibration material. Results for erythrocyte count and hemoglobin were accepted if within 2.7 percent and 2.6 percent, respectively, of the values assigned. Hematocrit was measured in duplicate by centrifugation (Sigma 2M, Christ, Osterode, Federal Republic of Germany) at 15,000 × G for 5 min using 75 mm borosilicate capillary tubes (Houmsen, Risskov, Denmark); the hematocrits were read with an Adams Micro-Hematocrit reader (Clay Adams, Becton-Dickinson, Parsippany, NJ) according to the NCCLS approved standard.[13] Mean cell volume and mean cell hemoglobin content were calculated from the erythrocyte count, hematocrit, and hemoglobin results.

Reticulocytes were counted in unfixed blood films following supravital staining with brilliant cresyl blue (1.5 g/L). Results are given as number of reticulocytes per 1000 erythrocytes.[14]

Serum iron (Fe^{3+}) was determined on a Prisma multichannel analyzer (New Clinicon, Stockholm, Sweden) at 600 nm after complex formation with 2,4,6-tri(2-pyridyl)-1,3,5-triazine following release from transferrin by hydrochloric acid and reduction by ascorbic acid.[15] We found a total coefficient of variation of 4 percent at an average of 31 μmol/L; the 95 percent reference interval using this method is 9–35 μmol/L.

Serum ferritin was measured by a sandwich radioimmunoassay (Bio-Rad, Richmond, CA) using an NE 1600 gammacounter (Nuclear Enterprises, Edinburgh, Scotland). The total coefficient of variation was 6.2 percent at 366 pmol/L, 6.1 percent at 64.6 pmol/L and 15.0 percent at 13.5 pmol/L; the lower limit of the 95 percent reference interval is given by the manufacturer as 44 pmol/L for males below 45 years and 100 pmol/L for males above that age.

Serum transferrin and haptoglobins were determined by immunoturbidimetry in a Cobas Fara centrifugal analyzer (Hoffmann-La Roche, Basle, Switzerland).[16] We used antibodies from Dakopatts (Copenhagen, Denmark). For transferrin, the total coefficient of variation was 3.1 and 2.4 percent at mean values of 32.2 and 49.1 μmol/L, respectively; the 95 percent reference interval using this method is 24.5–39.0 μmol/L. For haptoglobins, the total coefficient of variation was 3.4 and 3.9 percent at mean values of 1.16 g/L and 1.80 g/L, respectively; the 95 percent reference interval for healthy individuals is 0.35–2.40 g/L.

Results

Lead Exposure

All B-Pb levels at the battery factory (Table 1) were higher than those seen at the control facility, but only one was above 2.9 μmol/L, a legal limit used in several countries. However, the median was above a proposed health-based limit of 1.9 μmol/L.[4] The ZPP levels overlapped only slightly between the two groups (Table 1), thus confirming the increased lead exposure at the battery factory and the expected interference with the heme biosynthesis.

Hematological Status

The hematological parameters showed minimal differences between the two groups (Table 1). The lead-exposed workers showed a slightly lower average haptoglobin level, and somewhat lower reticulocyte counts than did the controls. A few individuals in both groups showed high ferritin levels. Thus, in general, the hematological parameters indicated essentially normal conditions. Most importantly, no difference was seen in iron status between the two groups.

Effects of Phlebotomy

On days 5 and 11, the hematological parameters reflected the recent blood loss in both groups. In particular, the reticulocyte counts began to increase. However, on both occasions, the lead-exposed workers showed a significantly lower number of reticulocytes, as compared to the controls (Figure 1). Otherwise, no significant differences were seen between the two groups.

On day 15 after blood donation, both groups still showed significantly decreased hemoglobin, hematocrit and erythro-

TABLE 1—Indicators of Lead Exposure and Hematological Parameters in 25 Lead-Exposed Workers and 25 Age-Matched Controls at the Time of Phlebotomy[a]

Indicators	Exposed Workers		Control Workers		P value
	Median	Range	Median	Range	
Blood lead (μmol/L)	2.14	0.93–3.59	0.35	0.13–0.84	0.000
ZPP (μmol/mol Hb)	108	34–429	29	13–51	0.000
Hemoglobin (mmol/L)	9.3	8.5–10.0	9.2	8.5–10.3	NS
Hematocrit ($\times 10^{-2}$)	44.5	41.0–48.0	44.7	41.0–47.5	NS
Erythrocytes ($\times 10^{12}$/L)	4.93	4.27–5.20	4.88	4.42–5.59	NS
Reticulocytes ($\times 10^{-3}$)[b]	7	2–27	10	0–18	NS
Mean cell volume (fl)	91	86–102	92	82–106	NS
Mean cell hemoglobin (fmol)	1.88	1.80–2.13	1.91	1.67–2.07	NS
Iron (μmol/L)	18	13–34	19	8–41	NS
Transferrin (μmol/L)	32.7	28.5–40.1	32.5	17.8–39.4	NS
Ferritin (pmol/L)	75	28–563	83	15–478	NS
Haptoglobins (g/L)	1.10	0.42–2.69	1.47	0.55–2.80	0.11

[a] Probability was calculated by Mann-Whitney U-test. NS denotes not significant.
[b] Number per 10^3 erythrocytes.

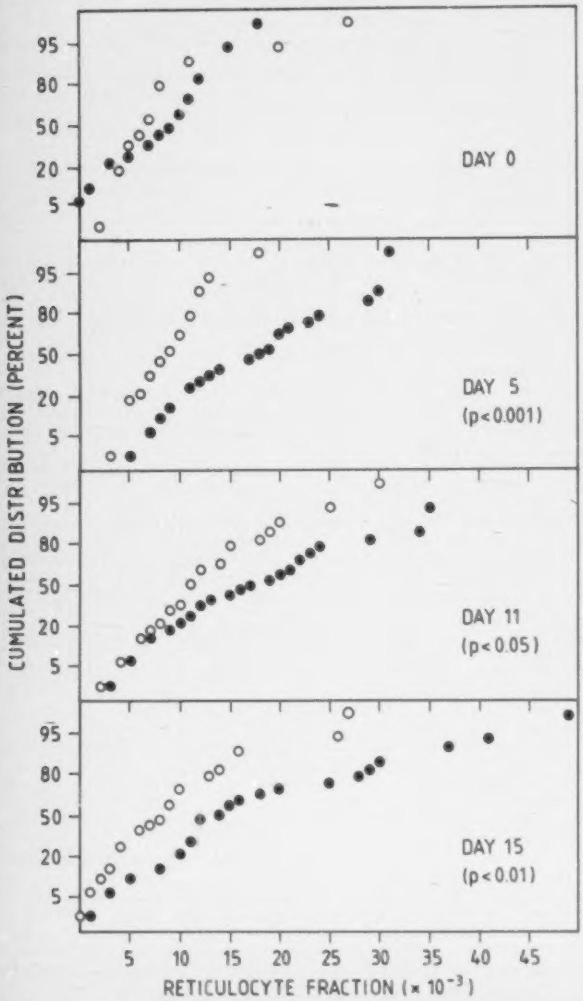

FIGURE 1—Cumulated Distribution (probability scale) of Reticulocyte Counts (per 1000 erythrocytes) at the Time of Phlebotomy (day 0), and 5, 11, and 15 Days Later (open circles, lead-exposed workmen; filled circles, age-matched controls).

cyte count, as compared to the day of phlebotomy. However, the decrease in hemoglobin and erythrocyte count was now significantly larger in the lead workers as compared to the control group ($p < 0.01$). As a further indication of the difference in blood regeneration activity, the increase in reticulocyte counts from day 0 to day 15 was less in the lead workers than in the controls (upper and lower panel of Figure 1).

When comparing the hematological data of the two groups at day 15, the high-lead group had significantly lower hemoglobin, and erythrocyte and reticulocyte counts, as compared to the controls (Table 2 and lower panel of Figure 1). No difference was seen in the hematocrit, mean cell volume, or mean cell hemoglobin.

TABLE 2—Hematological Parameters in 25 Lead-Exposed Workers and 25 Age-Matched Controls 15 Days after Phlebotomy[a]

	Exposed Workers		Control Workers		
	Median	Range	Median	Range	P value
Hemoglobin (mmol/L)	8.4	7.7–9.4	8.7	7.8–9.8	0.006
Hematocrit ($\times 10^{-2}$)	42.0	38.0–46.3	41.8	39.0–46.0	NS
Erythrocytes ($\times 10^{12}$/L)	4.38	4.04–4.77	4.58	4.11–5.23	0.020
Reticulocytes ($\times 10^{-3}$)[b]	9	0–27	14	1–49	0.003
Mean cell volume (fl)	93	82–106	92	84–101	NS
Mean cell hemoglobin (fmol)	1.89	1.82–2.11	1.92	1.72–2.15	NS

[a] Probability was calculated by Mann-Whitney U-test. NS denotes not significant.
[b] Number per 10^3 erythrocytes.

Discussion

The cross-sectional data at the time of phlebotomy confirmed previous findings[1-3] that hemoglobin levels are within normal limits in male workers exposed to lead below the legal B-Pb limit of 2.9 μmol/L. The slightly decreased haptoglobin levels could be related to a lowered erythrocyte lifespan, as decreases in this parameter most often reflect hemolysis or dyshemopoiesis.[17] No significant tendency toward lower mean cell volume was found in the lead-exposed workers, although anemia in lead poisoning may be of the microcytic type.[4,5]

The blood donation appeared to have similar effects on the two groups, except that the lead workers exhibited almost no increase in the reticulocyte count on day 5 and later lagged behind in reticulocyte counts. This deficient response to the blood loss is unexpected, because heavy lead exposure is known to cause an increase in reticulocyte counts, presumably as a result of the continuously increased demand on blood formation.[4,5]

Two weeks after phlebotomy, indications of increased blood formation should be apparent;[7-10] the findings in the control group concur with this pattern. However, the lower hemoglobin and erythrocyte count in the lead-exposed workers suggested delayed blood regeneration. No difference was detected in the hematocrit determination, perhaps due to the increased mean cell volume in the lead workers 15 days after phlebotomy. Although small, the differences in hemoglobin and erythrocyte count are considered valid, as comparability was ensured by vigorous quality control. Also, a delay in blood regeneration is in accordance with the slow increase in reticulocyte numbers seen in the lead workers.

Phlebotomy may be viewed as an artificial situation, but the half-liter blood loss is a reproducible intervention resulting in an increased demand on blood formation similar to, though probably less extensive than, the demand seen in a variety of medical conditions with excess blood loss. Also, this blood loss in blood donors represents an acceptable type of intervention that is known to cause no significant risk.

The present study of blood regeneration indicates that the reserve capacity was reduced in the lead workers despite the fact that minimal hematological changes were apparent before phlebotomy. In general, the level of erythropoiesis will depend on the marrow's capacity to proliferate, on erythropoietin stimulation, and on iron supply.[18,19] In the lead-exposed workers, the most important limiting factor in the rate of blood production after blood loss is likely to be the amount of available iron. As lead interferes with the utilization of iron in the bone marrow,[4,5] insufficient iron may be biochemically available for increased blood formation, despite a normal iron status. None of the subjects used iron supplements, and possible differences in dietary iron intake after blood donation would be unlikely to cause changes in iron status large enough to explain the differences in hemoglobin and erythrocyte count. The biochemical indications of deranged heme biosynthesis and of decreased red-cell survival seen in lead-exposed workers would therefore appear to have physiological significance.

The decision whether or not a chemically induced change has any health significance is often difficult. However, decrements in reserve capacity would generally be regarded an adverse effect that should be prevented. Wide agreement seems to prevail on this point, despite international differences in preventive strategies, as evidenced, for example, by occupational exposure limits. Thus, according to a report from the US National Academy of Sciences, a nonadverse effect of a chemical exposure must not result in impairment of functional capacity or the ability to compensate for additional stress.[20] Similarly, in the Soviet Union, effects are regarded adverse or abnormal if departures from the generally accepted "normal" values become manifest under functional or biochemical stress.[21-23] A World Health Organization study group also expressed the need to protect against decrements in homeostasis or decreases in functional capacity and therefore initiated an effort to recommend health-based limits for occupational exposures.[4]

Unfortunately, studies on reserve capacity are few, and this lack of information hampers the detailed evaluation of chemical hazards. Thus, the theoretical approaches to prevention should inspire appropriate experimental and epidemiological studies to explore more fully how chemical exposures may interfere with homeostatic and compensatory mechanisms. With the multicausal etiology of many diseases, such subclinical effects of numerous environmental and occupational exposures may well be of importance. The present study has illustrated the possible significance of this concept.

ACKNOWLEDGMENTS

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OSHA awards $1 million in Job Safety and Health Education Grants

Secretary of Labor Elizabeth Dole recently announced that grants totaling $1 million have been awarded to eight labor and four employer nonprofit associations to continue developing job safety and health training and education programs. Since this "New Directions" effort began in 1978, OSHA has awarded approximately $81 million in job safety and health grants to 202 organizations.

Information about the "New Directions" program is available from the Occupational Safety and Health Administration Training Institute, 1555 Times Drive, Des Plaines, IL 60018. Tel: (312) 297-4810.

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The Expense of Testing in a Teaching Hospital: The Predominant Role of High-Cost Tests

ROBERT M. HARTLEY, MD, MSC, MIRIAM A. MARKOWITZ, MSC, AND ANTHONY L. KOMAROFF, MD

Abstract: We analyzed all 32,206 tests ordered in a random sample of 1,000 patients admitted to a teaching hospital. The analysis examined costs instead of charges, included professional costs in addition to hospital costs, and considered the effect of combining tests that are ordered as a panel (e.g., cardiac enzymes) or as part of a procedure (e.g., bronchoscopy). High-cost tests (costing more than $100) accounted for only 4 percent of the total number of tests ordered, but for nearly 50 percent of total test costs. The pattern varied by clinical service: high-cost tests accounted for 31, 47, and 51 percents of total test costs on the obstetrics, medicine, and surgery services, respectively. It would seem to be efficient to concentrate on the use of high-cost tests in attempting to foster cost-effective use of diagnostic technologies. (*Am J Public Health* 1989; 79:1389–1391.)

Introduction

Many billions of dollars are spent each year on diagnostic technologies. How this money for diagnostic testing is spent remains unclear. While attention formerly focused on the use of high-cost tests, many have argued recently that the high volume ordering of inexpensive items may constitute the greatest fraction of total diagnostic testing costs.[1,2] Several reports have lent some support to this argument.[3,4] However, these studies have been designed to address other questions and have not attempted to measure directly the actual costs of testing.

Because of the importance of this issue in understanding how large sums of health care dollars are being spent and in choosing the best targets for cost-containment strategies, we have studied the cost of diagnostic testing in a sample of hospitalized patients. Because charges may poorly reflect true costs[5] and professional fees can add substantially to a test's cost, we have differentiated *costs* from *charges* and have examined both *hospital* and *professional* billing in our analysis.

Methods

Patient Sample

The Brigham and Women's Hospital in Boston, Massachusetts is a 720-bed tertiary hospital with an active teaching program. We randomly selected 1,000 patient admissions to the medical, surgical, gynecological and obstetrical services during fiscal year 1982. We included gynecological service patients with surgical patients since, in terms of resource consumption, they were more like surgical than obstetrical patients. Infants born in the hospital were excluded from the sampling frame. All data were obtained from and analyzed by a computerized system (CHASE System) built by one of us (AK), which stores itemized resource utilization data.

Definition of a Test

We defined a "diagnostic test" as any hospital procedure that was requested solely to seek information about a patient's status and which: required some sort of machinery; had been specifically requested in each instance of use; was not performed in a regular operating room; and did not involve continuous monitoring (e.g., central venous pressures) for an undefined time period. Using this definition, we examined all tests ordered in the sample of 1,000 patients: 1,195 different types of tests, ordered on 32,206 occasions.

We arbitrarily defined a high-cost test as one costing $100 or more, and a low-cost test as one costing less than $100.

Hospital Charges and Costs

We chose to estimate costs by two standard methods: ratios of costs-to-charges (RCCs) and relative-value units (RVUs). RVUs were either taken from College of American Pathologists standards, or developed through time-motion studies in our laboratories. RCCs were developed internally through detailed analyses of the laboratory's direct costs: the salaries of all personnel, including the physician laboratory director; supplies and equipment; and allocated expenses from all non-revenue producing cost centers, using a methodology that has been described in detail elsewhere.[6,7]

Analysis revealed no important differences when costs were estimated by the RCC or RVU method. Hereafter, all cost estimates are based upon RCC methodology.

Professional Charges and Costs

Because radiological, medical, and surgical professional groups billed separately for their services, we reviewed their billing data for each of our 1,000 patients. Pathology and cytology services did not bill separately for professional services. However, because professional labor constitutes a major proportion of these services' costs, we identified and separated the professional cost components. Routine hematology and chemistry professional costs were not separated because these professional costs contribute a very small proportion to their respective total test costs.

No reliable measures of true professional costs were available. Of available sources, the 1982 Medicare Part B customary reimbursements appeared to best express the direct costs of physician time and the indirect allocated costs of his or her business. However, because these estimates are likely in some cases to overestimate costs, we have conducted all analyses with and without professional costs

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included. Conclusions about the relative contribution of high-cost items were not substantially affected.

Determination of Unit Cost of Each Type of Test

The unit cost of each type of test included both the hospital cost and, if there was one, the professional cost. In addition we aggregated the costs of several tests when they were ordered simultaneously for the same clinical purpose.

Specifically, this was done in two instances: 1) *Test panels*: whenever several clinically-related tests (e.g., cardiac enzymes or electrolytes) were ordered on the same day; 2) *Procedures*: whenever a diagnostic procedure was performed which of necessity generated a number of different tests (e.g., a lumbar puncture which required measurements of cerebrospinal fluid glucose, protein, cell count, culture, and cytology) in order to complete the diagnostic intervention.

We examined the impact on our conclusions of these aggregations and found that test panels predominantly involved low cost tests which, even when aggregated, remained low-cost items under $100 and mostly under $10. The disaggregation of test panel-related and procedure-related tests did not materially change the ratio of high-cost to low-cost tests. Hence, all results reported involve aggregation of test panel- and procedure-related tests, a process which we believe make most clinical sense.

Results

Patient Sample

Our random sample of 1,000 patients was representative of the more than 25,000 patients (except newborns) admitted to Brigham and Women's Hospital between October 1, 1981 and September 30, 1982; age, sex, distribution by service, length of stay, and mean charges were virtually identical.

Volume and Total Costs

All Tests—For our sample of patients, physicians ordered 32,206 tests, which generated $763,108 in hospital charges, $554,811 in hospital costs, and $121,303 in professional costs. Test volume and costs, per patient-hospitalization, varied greatly across the services, with costs for an average medical admission being nearly 14-fold higher than for an average obstetrical admission (Table 1). The professional fee component of test costs averaged 12–19 percent of total test costs on each hospital service (Table 1).

Low-Cost and High-Cost Tests—Low-cost tests were ordered in high volume, accounting for 96 percent of the number of all tests ordered; 68 percent cost less than $10; 85 percent cost less than $20.

Total hospital test costs had two major peaks which occurred at the opposite ends of the distribution. The low-end peak was accounted for by the high-volume low-cost tests; non-angiographic radiology procedures dominate the mid-range ($100–500 each) and various angiographic investigations primarily account for the high-end peak (greater than $500 each), as shown in Figure 1.

TABLE 1—Test Usage and Costs Per Patient On Each Hospital Service

Hospital Services	Number of Tests	Hospital Test Costs	Professional Test Costs	Total Test Costs
Obstetrics	8.6	$ 85.72	$ 11.70	$ 97.42
Surgery & Gynecology	34.7	636.29	145.33	781.62
Medicine	62.4	1108.97	240.07	1349.04
Average for All Services	32.0	554.81	121.30	676.11

High-cost tests accounted for about 50 percent of total costs; tests costing more than $245 accounted for nearly 30 percent of total costs (Figure 2).

Professional Costs

Professional costs contributed 17.9 percent of total costs. The cumulative distribution of professional costs, plotted according to the total test cost with which it was associated, paralleled that of total costs (Figure 2). In fact, the majority of the professional costs were incurred by tests costing less than $60, indicating that the inclusion of professional costs did not cause high-cost items to become the major contributors to total test costs.

Differences: Medical, Surgical, and Obstetrical Services

Diagnostic testing costs accounted for 14 percent of total hospital costs (professional costs excluded) in the total sample of 1,000 patients: 20 percent on the medical service, 12 percent on the surgical service, and 5 percent on the obstetrical service.

In surgery and medicine, expensive tests made the predominant contribution. High-cost tests accounted for 31, 47, and 51 percent of total test costs on the obstetrical, medical and surgical services, respectively (Figure 3). Tests costing more than $450 accounted for about 20 percent of total test costs for medical and surgical patients but none of the costs for obstetrical patients.

Discussion

Diagnostic testing makes a large contribution to the costs of care for hospitalized patients. Our results indicate that tests costing $100 or more account for one-half of the total diagnostic costs. We made the inherently arbitrary decision to define unit test costs of $100 or greater as "high-cost" because the distribution of total test costs by unit costs (Figure 1) demonstrated a natural break point at this level. The next natural break point occurred at $175, a substantially greater unit cost, and the unit prices for such tests were typically well over $200; it did not seem that most people would use the phrase "low cost" to apply to such tests. It is especially interesting to note the very large contribution to total test costs by tests whose unit costs were greater than $500 (Figure 1).

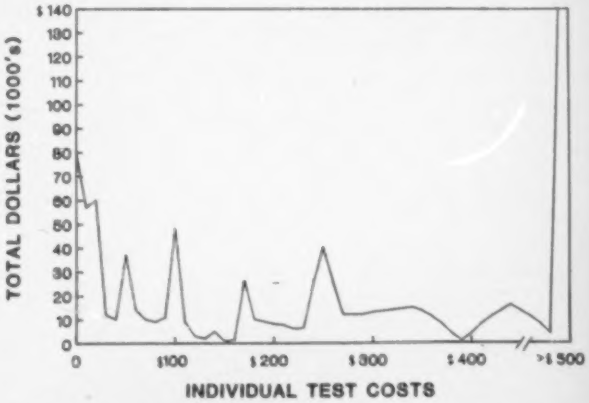

FIGURE 1—Total Test Costs, by Unit Cost of Each Type of Test Ordered, for 1,000 Randomly Selected Patients, 1982.

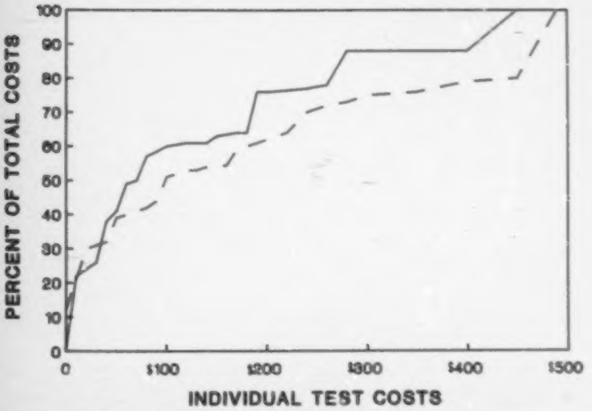

FIGURE 2—Cumulative Distribution of Total Test Costs, and of Professional Costs, by Unit Cost of Each Type of Test Ordered, for all Hospital Resources Utilized, in 1,000 Randomly Selected Patients, 1982. KEY = Solid line is professional cost. Broken line is total test cost.

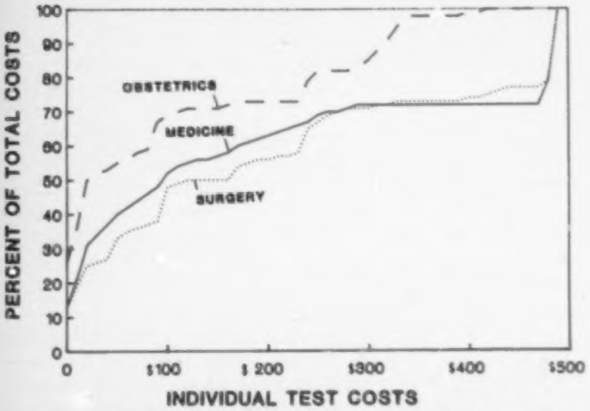

FIGURE 3—Cumulative Distribution of Total Test Costs, by Unit Cost of Each Type of Test Ordered, for each Hospital Resource Utilized, in 1,000 Randomly Selected Patients, 1982.

We believe the distinction we have made between high-cost and low-cost tests is useful for several reasons. First, attempts at control of the use of most high-cost items have traditionally employed regulatory measures directed at the capital expenditures needed for test machinery, an approach of no value for modifying low-cost testing. Second, reported experience suggests that at least one cost containment strategy, organized educational programs, may be less effective in reducing the use of high-cost than low-cost technologies.[4,8] Finally, test-ordering habits vary with test cost. Low-cost tests are often repetitively or reflexly ordered before information is available as to whether they are needed, whereas higher cost tests are usually sequentially ordered depending upon results from preceding investigations.

Our conclusions about the dominant contribution of high-cost items were unaffected by the inclusion or exclusion of professional costs. However, the contribution of professional costs to total test costs is nevertheless substantial. In our study, it represented more than 20 percent of the total for medical and surgical patients. In those situations where the test-ordering physician is the same as the test-performing physician—such as endoscopy or various cardiology procedures—the magnitude of the professional reimbursement represents an incentive for ordering and thus an additional challenge for cost-containment attempts.

Several aspects of our methodology should be noted. Our hospitalized patients were in a teaching center. In such a setting, the test-ordering physician is relatively inexperienced; there is easy access to costly test machinery and procedures; and patients may be more severely ill.[9] For these reasons, our findings may not be generalizable to non-teaching hospitals. On the other hand, we may have underestimated the contribution of high-cost tests, even at a teaching hospital: we did not include as high-cost items several *therapeutic* procedures which have diagnostic components (e.g., laparoscopy, arthroscopy).

While low-cost tests may contribute more in other settings, our results indicate that high-cost tests make an important—in this study, predominant—contribution to the costs of diagnostic testing. Cost containment efforts should target strategies designed to ensure effective use of high-cost tests.

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The Mortality Consequences of Raising the Speed Limit to 65 MPH on Rural Interstates

HERBERT M. BAUM, PHD, ADRIAN K. LUND, PHD, AND JOANN K. WELLS, BA

Abstract: As of April 1987, states were permitted to raise the speed limit on rural interstates to 65 mph without incurring federal sanctions; 38 states elected to do so in 1987. Fatality data for the months when the new limit was in effect in 1987 were compared with fatalities in the same months of 1982–86 on rural interstates and other rural roads. Fatalities on rural interstates in the states with increased speed limits in 1987 were conservatively estimated to be 15 percent higher than they would have been if the states had retained the 55 mph limit (95% CI = 6, 24). Among states that retained the 55 mph limit, fatalities on rural interstates were 6 percent lower than expected (95% CI = −23, 13). (*Am J Public Health* 1989; 79:1392–1395.)

Introduction

Speed limit laws, which date back to 1901, have traditionally been the responsibility of the states. However, in 1974, Congress responded to the oil embargo by directing the US Department of Transportation to withhold highway funding from states that did not adopt the national maximum speed limit of 55 mph (PL 92-239 and 93-643).

The National Research Council (NRC) attributed 4,000 fewer fatalities from 1973 to 1974 to the decreased speeds. Despite some increases in travel speeds since 1974, the NRC concluded that in 1983 the 55 mph speed limit was responsible for approximately 3,000 fewer fatalities, 3,500 fewer serious, severe, and critical injuries, and 47,000 fewer minor and moderate injuries.[1] The NRC estimated that returning the speed limit on rural interstates to their pre-1974 level in all states would result in 500 more fatalities annually, a 20–25 percent increase on these highways.[1]

In 1987, Congress enacted the Surface Transportation and Uniform Relocation Act (PL 100–17), which allowed states to increase the speed limits on rural interstate highways up to 65 mph. By the end of 1987, 38 states had raised their speed limits on rural interstates to 65 mph. Two additional states, Georgia and Virginia, followed suit in 1988. The District of Columbia, Delaware, and Alaska had no eligible miles of highway, leaving eight states with eligible mileage that have not increased speed limits (Figure 1). In December 1987, Congress established a four-year demonstration project that expanded the list of eligible highways to include turnpikes and other highways that meet federal interstate standards. Consequently, Alaska increased the speed limits to 60 mph on some roads, and some other states raised the speed limit on non-interstate rural roads.

Recent observational studies in several states found that, although the previous 55 mph speed limits had been exceeded frequently, the increased speed limits resulted in more motorists traveling at faster speeds.[2] In New Mexico, the percentage of passenger cars exceeding 65 mph went from 37 percent in the first month of implementation to 61 percent in the eighth month; the percentage of passenger cars exceeding 70 mph quadrupled, from 5 percent to 21 percent. In Virginia, the percentage of passenger cars exceeding 65 mph went from 26 percent immediately before the change to 54 percent immediately after, and the percentage exceeding 70 mph went from 7 to 15 percent.[3]

The National Highway Safety Administration (NHTSA) issued a report in January 1989 on the effects of the higher speed limit. NHTSA noted that it was too early to assess the long-term impact of the new speed limit on highway safety. However, the most sophisticated of the report's analyses (a regression model that controlled for fatalities on other roads, and the change in rural interstate mileage, for each year since 1975) indicated that rural interstate fatalities were 16 percent higher than expected.[4]

An American Automobile Association (AAA) sponsored analysis of the effect of the 65 mph limits in Indiana showed no effect on fatalities.[5] However, individual state analyses can often be misleading because the number of crashes on rural interstates are few and the statistical variation associated with these counts can be quite large. In Indiana, for example, there are about 90 fatalities annually on rural interstates. By chance, this count would vary by as much as 20 percent (the standard error of a Poisson distribution with a mean of 90 is ± 9.5). Further, one month after the speed limit was raised, Indiana's mandatory seat belt use law went into effect, and the AAA analysis did not adequately address this change.

This paper presents analyses of fatalities on highways with increased speed limits for the 38 states that raised limits in 1987. By including all these states, the analysis minimizes the problem of statistical unreliability inherent in individual state analyses. In addition, to help control for other contemporaneous changes that might affect fatal crashes on rural interstates, the analysis also examined the fatal crash experience on other roads that might be expected to respond

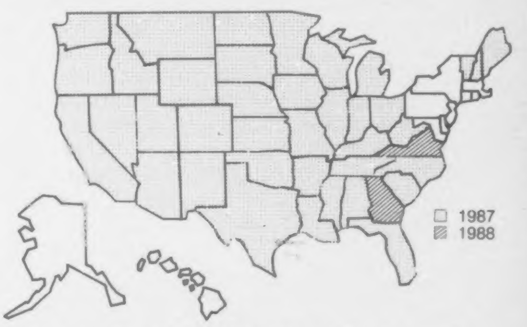

FIGURE 1—States with 65 MPH Speed Limits on Rural Interstates

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similarly to many of these changes. Fatality data for the months of higher limits in 1987 were compared to the average for the same months during the previous five years.

Method

Fatality data are from the Fatal Accident Reporting System (FARS), the federal data base of detailed reports on all fatal motor vehicle crashes on public roads in the United States.[6] States enacted the higher speed limits on various dates throughout 1987 (Table 1). As shown in the table, fatalities on rural interstates in 1987 were counted for each state for all months following the month in which the speed limit was raised to 65 mph. For example, in Alabama where the increased limit became effective on July 20, fatalities were included for August through December. The month in which the change occurred was excluded because of possible delays in posting signs. Rural interstate fatality counts were also obtained for the same calendar months in each of the previous five years, 1982–86. Both overall fatality counts and counts of fatalities among occupants of passenger vehicles (cars, light trucks, and vans) were examined.

Under the 1987 law, a state can post a 65 mph limit on a "highway on the interstate system located outside of an urbanized area of 50,000 or more persons." This definition of eligible highways does not correspond exactly with the highway designations used in FARS; all interstates in FARS that are coded as rural meet the Act's definition, but a small percentage of FARS designated urban highways also meet the definition. However, because 96 percent of the interstate highway miles eligible to be posted at 65 mph are rural interstates as defined by FARS, and 97 percent of these eligible miles were posted at higher limits, these highways were used to represent those with 65 mph limits.[4] An alternative way to count fatalities was to tabulate using the actual posted speed limit recorded in FARS to indicate eligible highways; however, this approach would have precluded comparisons prior to 1987.

TABLE 1—Effective Dates for 65 mph Speed Limit and Months of Data Included in Analysis, 38 States

State	1987 Effective Date	Analysis Months
Alabama	July 20	August–December
Arizona	April 15	May–December
Arkansas	April 20	May–December
California	May 28	June–December
Colorado	April 6–10	May–December
Florida	April 27	May–December
Idaho	May 2	June–December
Illinois	April 27	May–December
Indiana	June 1	July–December
Iowa	May 12	June–December
Kansas	May 14	June–December
Kentucky	June 8	July–December
Louisiana	April 8	May–December
Maine	June 17	July–December
Michigan	November 29	December
Minnesota	June 17	July–December
Mississippi	April 14	May–December
Missouri	May 1	June–December
Montana	April 16	May–December
Nebraska	April 27	May–December
Nevada	April 13	May–December
New Hampshire	April 16	May–December
New Mexico	April 2	May–December
North Carolina	August 10	September–December
North Dakota	April 16	May–December
Ohio	July 15	August–December
Oklahoma	April 6	May–December
Oregon	September 27	October–December
South Carolina	July 15	August–December
South Dakota	April 15	May–December
Tennessee	May 8	June–December
Texas	May 9	June–December
Utah	May 21	June–December
Vermont	April 21	May–December
Washington	April 20	May–December
West Virginia	April 20	May–December
Wisconsin	July 17	August–December
Wyoming	May 19	June–December

Because motor vehicle crash fatalities fluctuate from year to year for a variety of known and unknown reasons, changes observed on rural interstates might be due to other trends in motor vehicle crashes. Therefore, fatality counts on rural interstates were compared to those on all other rural roads.

The odds that a fatality occurred on a rural interstate road (versus all other roads or all other rural roads) were compared to the same odds averaged over the previous five years (1982–86). The 95 percent confidence limits for this odds ratio were computed by methods given in Fleiss.[7]

The fatality experience of states that increased the speed limit was not compared directly to the experience of states that retained the 55 mph limit. As shown in Figure 1, most states that retained the 55 mph limit are in the densely populated northeast and differ on a number of factors that could affect traffic fatalities. In addition, the fatality experience of these states may have been affected by the spillover of higher speeds from states that changed, thereby making a direct comparison inappropriate. Nevertheless, the fatality experience of the states that retained 55 mph limits was examined for any evidence that changes in the 65 mph states were simply part of a larger national trend. Odds ratios, similar to those for the 38-state analysis, were constructed using data from eight states (Connecticut, Georgia, Maryland, Massachusetts, New Jersey, New York, Pennsylvania, and Virginia) that had 55 mph limits and at least 25 miles of rural interstate highway. Fatalities were counted only during the months of May through December to simulate a speed limit change in April of 1987. If there were no larger national trend in rural interstate fatalities in 1987, this odds ratio would be expected to equal unity.

Results

The numbers of fatalities on rural interstates during the months with higher speed limits in 1987 and the same months in 1982–86 for the 38 states with changed limits are shown in Figure 2. This figure shows that fatalities on rural interstates posted with 65 mph limits in 1987 were considerably higher than in any of the previous five years.

Although the number of fatalities on rural interstates increased slightly over the five baseline years, a similar trend was also found for the control series of fatality counts on other rural roads. The yearly odds of fatalities on rural interstates compared to other rural roads remained fairly constant until 1987. Specifically, for 1982 through 1986, the ratios were 0.097, 0.106, 0.109, 0.107, and 0.100, respectively.

There were 19 percent more fatalities on rural interstates in 1987 than the average for the previous five years, while there were only 4 percent more fatalities on other rural roads. For all fatalities, the odds of death on rural interstates versus other rural roads were 15 percent (95% CI = 6, 24) greater in

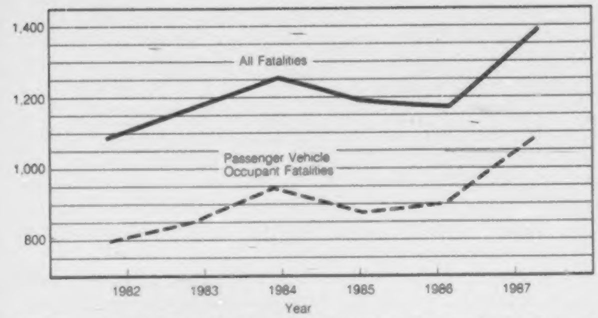

FIGURE 2—Fatalities on Rural Interstates during Months of Higher Speed Limits in 1987 and Same Months in 1982–86

1987 than the average for 1982–86 (Table 2). If *all* other roads are used for the comparison, the ratio yields a 16 percent effect.

The odds ratio for passenger vehicle occupant deaths showed a similar increase—16 percent (95% CI = 6, 28). There were 25 percent more passenger vehicle occupant fatalities on rural interstates in 1987 than the average of the previous five years, while on other rural roads the increase was only 8 percent. When all other roads were used as the comparison group, the odds for passenger vehicle occupant fatalities on rural interstates were 17 percent higher than expected.

A number of other factors that might affect the relation between speed limit changes and fatalities were also examined. These factors included different speed limits for cars and trucks, mandatory seat belt laws, daytime (6 am–9 pm) versus nighttime crashes, and the proportion of single or multiple vehicle fatal crashes. These analyses were done by subdividing the data for rural interstates and other rural roads for all fatalities by the appropriate factor and comparing the odds ratios. The estimated effect of 14 percent for the eight states with different speed limits for cars and trucks (California, Illinois, Michigan, Missouri, Ohio, Oregon, Texas, and Washington) was similar to the 15 percent effect for states with uniform limits. The effects were also similar in states with belt use laws (15 percent) and without belt laws (13 percent). For daytime and nighttime crashes, the effects were also similar (16 percent and 14 percent). The effect for fatalities involving single vehicles was 17 percent, and for fatalities involving multiple vehicles it was 10 percent. A test for homogeneity of the odds ratios was not significant implying no difference between the odds ratios.

The analysis for the eight states that retained 55 mph limits in 1987 indicated no effect during the comparison period. There was a 4 percent increase in all fatalities on rural interstates, but this was offset by an even larger increase on other rural roads (12 percent). The estimated odds ratio was 0.94 (95% CI = 0.77, 1.13).

Discussion

In the 38 states that set higher speed limits in 1987, fatalities on rural interstates were conservatively estimated to be 15 percent greater than they would have been if the states had retained the 55 mph limit on these roads. Increased fatalities were seen in 24 of the 38 states. In human terms, there were nearly 200 additional fatalities in 1987 during the months when the higher speed limits were in effect. This number will be larger for 1988 because two additional states have raised their speed limits, more miles of highways were eligible, and states will have had a whole year of experience.

In a 1988 interim report on the effect of the higher speed limit, NHTSA had expressed concern that the observed 1987 increase in fatalities began in the months prior to initiation of the higher limits.[8] To test this possibility, an alternative analysis examined fatalities for January through March of 1987 versus those for 1982–86. The odds ratio for fatalities on rural interstates versus other rural roads indicated that fatalities on rural interstates were about 6 percent higher than expected during the first quarter of 1987. Although the odds ratios in Table 2 could be adjusted for this first quarter increase, the odds ratio for the first quarter included 1.0 suggesting that the observed difference was due to random fluctuation. In addition, fatalities during winter months tend to fluctuate greatly from year to year because of varying weather patterns, and the current analysis could not control for these patterns. Moreover, there is the possibility that some drivers began increasing their travel speeds even before the speed limits were raised. Such anticipatory speed increases could have been triggered by news reports and publicity regarding possible federal action in early February and again in March.[9,10] For these reasons, it would have been inappropriate to adjust the estimated effects of the higher speed limits for the nonsignificant differences observed in January through March.

The estimated 15 percent increase in these data is somewhat smaller than the 20–25 percent increase predicted by the NRC partly because of the NRC assumption that all states would return to pre-1974 speed limits, often 70 and 75 mph. Because the energy associated with a crash increases exponentially with the speed of the crash, the likelihood of serious injury and fatality increases very rapidly as travel speeds increase in this range.[11] Therefore, even larger effects

TABLE 2—Number of Fatalities by Road Type and Time Period for All Fatalities and Passenger Vehicle Occupants

	Average 1987 Number	Average 1982–86 Number	Chi-Square Statistic	Odds Ratio	(95% Confidence Limit)
All Fatalities					
Rural Interstates	1,393.0	1,169.2			—
vs Other Rural Roads	11,713.0	11,259.2	10.40	1.15	(1.06, 1.24)
vs All Other Roads	19,607.0	19,110.2	13.20	1.16	(1.07, 1.26)
Passenger Vehicle Occupants					
Rural Interstates	1,084.0	864.8			
vs Other Rural Roads	9,321.0	8,632.4	9.53	1.16	(1.06, 1.28)
vs All Other Roads	13,906.0	13,031.8	11.50	1.17	(1.07, 1.29)

than estimated here would have been expected if states had returned to these higher limits.

In addition, the 15 percent increase in fatalities found here probably underestimates the true effect of the 65 mph limits. The effect was measured by comparing the odds of a fatality on rural interstates to other roads. If higher travel speeds on rural interstates spill over onto some of these roads, as suggested by research on speed adaptation, then the number of fatalities would have increased on these other roads as well.[12–14] Such a spillover would reduce the estimated effect (but not the true effect) of the higher speed limits as measured in this analysis.

The NHTSA report highlighted two factors that argued against a large increase in fatalities attributable to the 65 mph speed limits: the states that did not increase their speed limits also had an increase in fatalities on rural interstates and six states accounted for 64 percent of the excess fatalities in 1987.[4] The current analysis examined the odds ratio for the unchanged states for 1982–86 versus 1987 and found rural interstate fatalities were marginally fewer than expected for May–December of 1987 in states that retained 55 mph speed limits. Thus, there is no evidence of a larger national trend toward more fatalities on rural interstates in 1987.

When odds ratios were computed for each state, they were greater than unity in 24 states. Therefore, despite the dominance in the absolute numbers by six states, the trend of higher crash fatalities on rural interstates than on other rural roads was observed in more than three of five states that increased their speed limits. The fact that rural interstate fatality counts were not higher than expected in a minority of states is consistent with the statistical uncertainty associated with analyses of data from individual states.

In summary, the results of this analysis show that the 65 mph limits increased rural interstate fatalities in 1987 by approximately 15 percent, there was no evidence of a similar trend in states that retained 55 mph limits, and the increase was not limited to only a few states.

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ISHIB Calls for Abstracts on Hypertension in Blacks

The 5th International Interdisciplinary Conference on Hypertension in Blacks has issued a call for abstracts for its conference, to be held May 3–7, 1990 in Long Beach, California. The announced theme for the meeting is: "A Focus on Ethnicity: Negroids, Caucasoids, Mongoloids." The deadline for abstract submission is November 1, 1989.

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Homicide, Suicide, Motor Vehicle Crash, and Fall Mortality: United States' Experience in Comparative Perspective

IAN R.H. ROCKETT, PHD, MPH, AND GORDON S. SMITH, MB, CHB, MPH

Abstract: US mortality data on motor vehicle crashes, falls, suicide, and homicide for 1980 are compared with corresponding data for France, Japan, West Germany, and the United Kingdom. Unadjusted and age-specific death rates are presented, together with age-adjusted rates of years of life lost (YLL). A large male excess in rates is typical outside the fall category. Motor vehicle crashes are the predominant cause of YLL, and the United States manifests the highest YLL rates for each sex. US fall death rates at the older ages are exceeded by those of France and West Germany. The elderly generally manifest the greatest risk of suicide; American females exhibit a unique rate decline after ages 45–54 years, however. Beyond early adulthood, US suicide rates are lower than those of France, Japan, and West Germany. US homicide rates dwarf those of the comparison countries with 16- to 29-fold differentials separating prime-risk American males aged 25–34 years from their foreign counterparts. (*Am J Public Health* 1989; 79:1396–1400.)

Introduction

In a foreword to an influential report from the National Academy of Sciences, William Foege remarked on the persistence of injuries as a "major determinant of longevity" in the United States, and globally.[1] The 1980s have furnished much basic data on injury occurrence and witnessed renewed interest in their prevention.[2–7] Federal Government support has been strengthened by the formation of a Center for Environmental Health and Injury Control at the Centers for Disease Control, the establishment of five regional university-based injury prevention research centers, and growing sponsorship of pertinent research projects and prevention programs by the National Institutes of Health.[8,9]

Nationally, injuries rank fourth as a cause of death behind heart disease, cancer, and cerebrovascular disease,[10] but they are the premier killer of Americans between ages one and 45 years,[10] and are the leading cause of years of life lost (YLL) before age 65.[11] An international study revealed marked differences in all-cause injury death and age-adjusted YLL rates for the United States, France, Japan, the United Kingdom, and West Germany.[12] The aim of the present paper is to shed light on these findings through an examination of data on unintentional motor vehicular and fall mortality, suicide and homicide—the leading causes of injury mortality in the United States.[4] The four comparison countries are of particular interest due to similarities they share with the United States in terms of development and mode of government, and because of their close political and economic links to this country. All five countries have populations exceeding 55 million, and thus may be considered demographically large.

Methods

Data are adapted from 1980 age-, sex-, and cause-specific mortality tabulations published by the World Health Organization (WHO).[13] Total injury deaths (E800–E999) and deaths from motor vehicle traffic crashes (E810–E819), falls (E880–E888), suicide (E950–E959) and homicide (E960–E969) are classified under an abbreviated three-digit external cause code according to the basic tabulations list of the ninth revision of the *International Classification of Diseases* (ICD-9).[14]

YLL rates are adjusted for age by means of a direct standardization procedure,[15] using the 1980 US population as the reference. YLL represent the summation of the products of the number of deaths at each age, here starting at age one, and the years of life remaining up to the age ceiling, here age 65. Since the WHO age data are abridged, age at death for decedents is estimated as the mid-point of the respective age groups.

Results

Crude Rates and Causal Components

France manifests the highest crude injury death rates for either sex (116 per 100,000 males and 73 per 100,000 females) (Table 1). The United States has the second highest rate for males (105 per 100,000), West Germany the second highest rate for females. Male rates surpass female rates in all five countries, and the magnitude of the male excess ranges from 50 percent in the United Kingdom to 180 percent in the United States.

Motor vehicle crashes, falls, suicide, and homicide collectively account for between two-thirds and four-fifths of total injury deaths in each country. However, homicide is a major cause of death only in the United States. Homicide rates in the remaining four countries approach individual mortality rates for fire, poisoning, drowning, and other transportation (data not shown).

Except in Japan, motor vehicle crashes are the leading cause of injury mortality among males, with the United States having the highest sex-specific rates. Motor vehicle crashes are also the leading cause of injury mortality among females in the United States, but in the remaining countries they are surpassed by suicide, falls, or both. Male predominance universally characterizes motor vehicular mortality and suicide, but the sex differential is sharply reversed with respect to fall mortality in France, West Germany, and the United Kingdom.

Age-Specific Rates

Both sexes exhibit a bimodal age-specific pattern of motor vehicular mortality, irrespective of country (Figure 1). Peaks occur at ages 15–24 years and over 74, with male rates greatly exceeding female rates. Japan and the United King-

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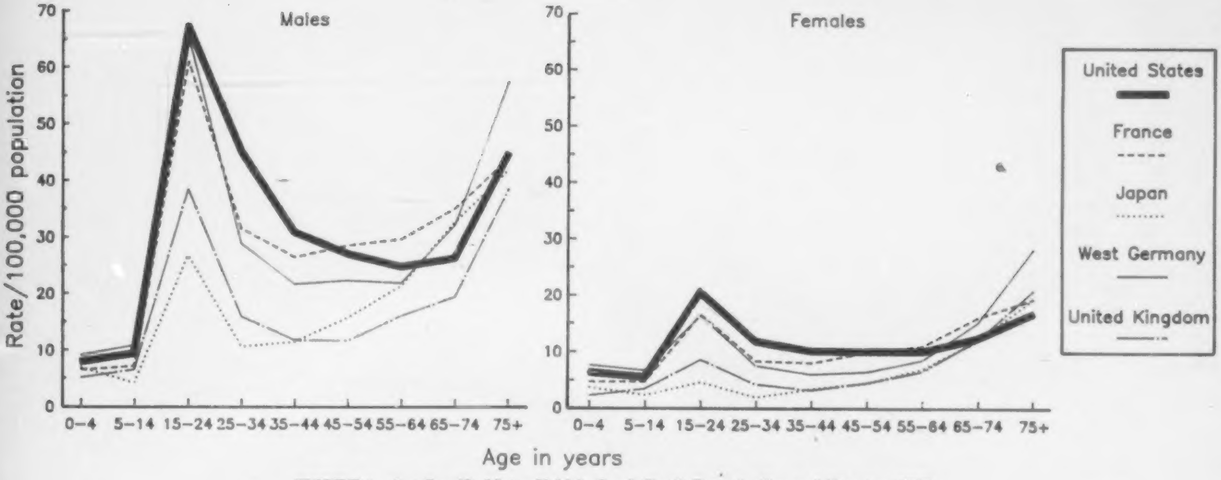

FIGURE 1—Age-Specific Motor Vehicle Crash Death Rates by Sex and Country, 1980

dom manifest the lowest motor vehicular death rates for both sexes, although there is a trend toward convergence at the upper end of the age spectrum. The United States has the highest motor vehicular death rate for females from ages 15 through 44 years, and the lowest rate in the over 74 population.

Fall mortality is profoundly selective of the elderly (Figure 2). In the over 74 age group, which reflects the greatest risk, there is considerable cross-national variation by sex, with rates peaking among French and West Germans. In France, West Germany, and the United Kingdom, female rates are approximately 50 percent higher than corresponding male rates. Japan has by far the lowest national rates, and US rates also are relatively low.

Generally, age-specific suicide rates are highest in France, Japan, and West Germany, with the elderly most at risk in all countries (Figure 3). Male rates universally exceed female ones. American females manifest a unique decline in their suicide rate after ages 45–54.

Homicide rates are usually lower than suicide rates, except in the two youngest age groups, where there is a major departure from the norm by the United States because of extremely high homicide rates among adolescents and younger adults of both sexes (Figure 4). In the prime-risk 25–34 age group, the US rate for males ranges from 16 to 29 times other national rates. The US rate for females in that age group, while less than one-fourth that for US males, is still more than triple the second highest national male homicide rate, that of West Germany.

Years of Life Lost

Motor vehicular, suicide, and fall YLL rates are invariably much greater for males than females (Table 2). This finding is repeated with US homicide YLL rates, which for each sex marginally surpass corresponding suicide rates, and dwarf other national homicide YLL rates. Suicide produces the highest sex- and cause-specific YLL rates in Japan; elsewhere it is motor vehicle crashes. Male suicide YLL rates are similar in the United States, France, Japan, and West Germany, and are at least double the British rate. Female suicide YLL rates are highest in France, Japan and West

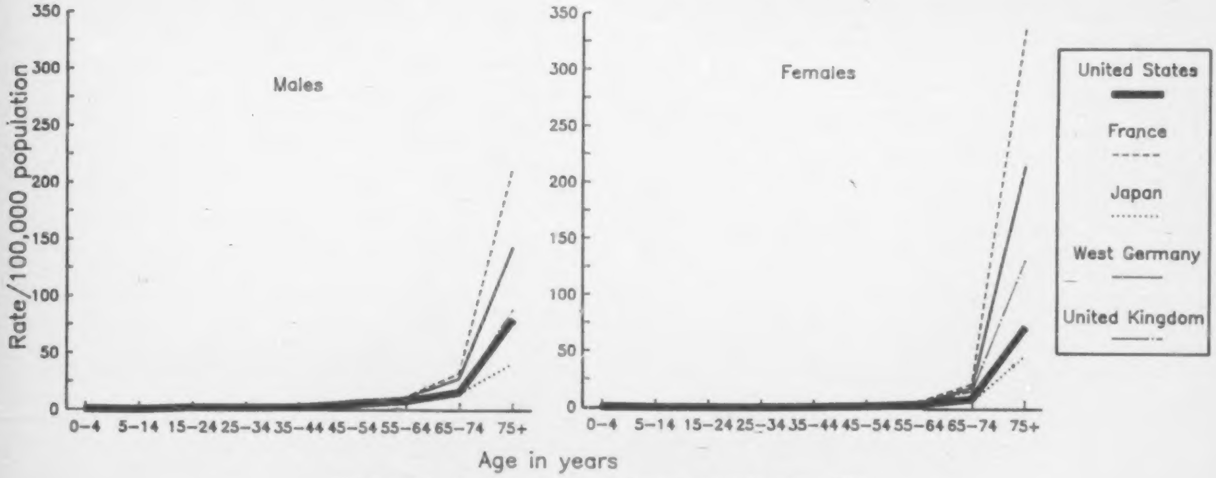

FIGURE 2—Age-Specific Fall Death Rates by Sex and Country, 1980

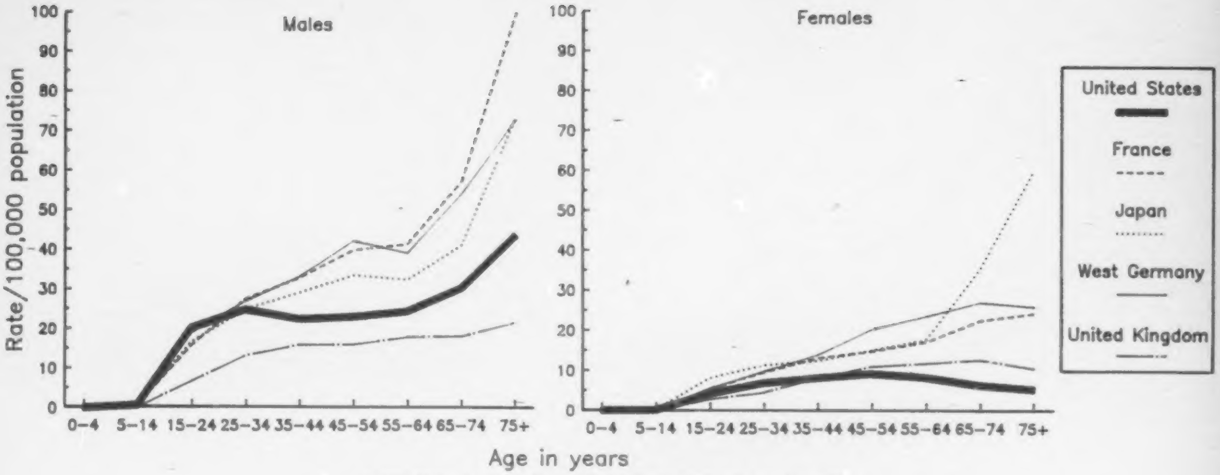

FIGURE 3—Age-Specific Suicide Rates by Sex and Country, 1980

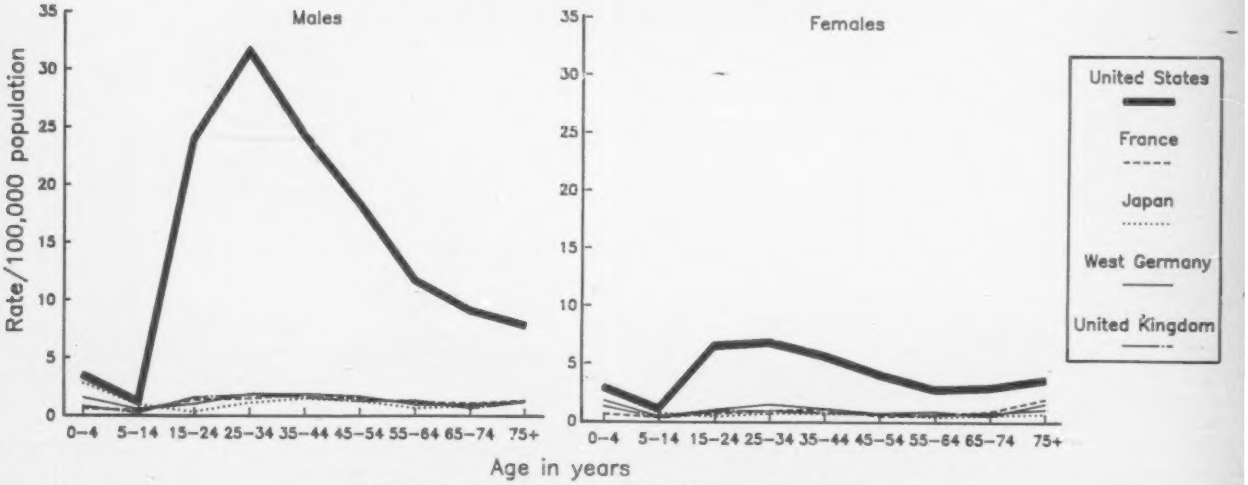

FIGURE 4—Age-Specific Homicide Rates by Sex and Country, 1980

Germany, and approach the British rate for males. Fall YLL rates are consistently small by sex and across countries.

Discussion

Focusing on 1980 motor vehicle crash, fall, suicide, and homicide data, this research has pointed to substantial differences in the injury mortality profiles of the United States and four other major Western bloc countries: France, Japan, the United Kingdom, and West Germany. Motor vehicle crashes are the predominant cause of injury mortality in the United States, and are also the predominant cause of YLL between ages one and 65 years in all countries under review, except Japan. The sharp peaks in motor vehicular death rates, which are exhibited at ages 15–24 years, have also been observed in many other countries.[16]

A better single indicator of road death risk than an overall population-based mortality rate is number of deaths per motor vehicle distance traveled. In 1980, the United States and the United Kingdom both registered 2.1 road deaths per 100 million vehicle kilometers traveled.[17] The Japanese figure was marginally higher (2.25). In contrast, the French and West German figures were 4.6 and 3.8, respectively. All countries in the study manifested appreciable declines on the exposure measure between 1970 and 1980; from 68 percent for Japan to 30 percent for the United States.[17,18] Similar declines occurred in the overall motor vehicular mortality rates.[13,19] A more protracted downward trend has also been noted for select populations.[20]

A recent US study revealed large inter-county differences in motor vehicle crash mortality rates, which varied inversely with population density and income.[21] The authors speculated that this variation reflected differences in a host of underlying variables, including road quality and design, speed, vehicle type and condition, seat belt use, and access to emergency care. These and other variables, such as alcohol use,[5] vehicle safety design features,[3] access to safer modes of transport, such as trains,[5] and seat belt legislation and related timing, compliance, and enforcement[22] should be integral to analytic epidemiologic research aimed at addressing spatial and temporal variations in the road toll.

TABLE 1—Injury Mortality by Sex, Cause and Country: Rates and Percentage Distribution, 1980*

Sex/Cause	United States		France		Japan		West Germany		United Kingdom	
Male	Rate	%	Rate	%	Rate	%	Rate	%	Rate	%
Motor Vehicle	34.4	32.6	30.5	26.3	15.1	24.2	30.4	34.7	17.6	33.7
Fall	6.4	6.1	15.0	12.9	5.1	8.2	11.6	13.2	6.9	13.2
Suicide	18.6	17.6	28.0	24.2	22.2	35.5	28.3	32.3	11.0	21.1
Homicide	17.0	16.1	1.2	1.0	1.1	1.8	1.3	1.5	1.2	2.3
Other Injury	29.1	27.6	41.2	35.6	19.0	30.3	16.0	18.3	15.5	29.7
All Injury	105.5	100.0	115.9	100.0	62.5	100.0	87.6	100.0	52.2	100.0
Female										
Motor Vehicle	12.0	31.7	10.8	14.8	4.8	16.9	11.0	21.1	6.9	19.4
Fall	5.4	14.3	30.0	41.1	2.5	8.8	19.3	37.1	12.1	34.0
Suicide	5.4	14.3	11.1	15.2	13.1	46.1	14.1	27.1	6.7	18.8
Homicide	4.4	11.6	0.8	1.1	0.8	2.8	1.0	1.9	0.8	2.2
Other Injury	10.6	28.1	20.3	27.8	7.2	25.4	6.6	12.8	9.1	25.6
All Injury	37.8	100.0	73.0	100.0	28.4	100.0	52.0	100.0	35.6	100.0

*Rates per 100,000 population.

Fall mortality primarily strikes the elderly. Yet geriatric fall deaths are probably markedly undercounted because many of the victims die of complications stemming from the initial injury, and may have a consequential condition coded as their underlying cause of death.[23–25] It is unknown how much of this contributes to the differences observed in this study. There has been a secular decline in the US fall death rate, with a reduction of 35 percent occurring between 1970 and 1980 alone.[5] This trend may be a product of improved medical care, such as hip surgery and earlier mobilization after hip fracture.[4,5] But also unknown is how much of the observed cross-national fall mortality differentials can be explained by quality of care considerations.

Both validation of the WHO cause-of-death data and estimation of the amount of variation in rates attributable to different national coding practices lie outside the scope of this study. However, an explanation was sought for the relatively high percentage of unintentional injury deaths among French males in 1980 that were classified by WHO under the rubric of "all other accidents," and were reflected in the residual injury mortality categories in Tables 1 and 2. The French age mortality distribution for "other" unintentional injuries corresponds to the distribution for motor vehicular injuries, probably due to a frequent tendency by French physicians to write "accident" (coded as unspecified unintentional injury) on the death certificate as the sole external cause descriptor in a fatal motor vehicular trauma case.*

Like motor vehicular death rates, suicide rates vary dramatically by country, age, and sex. The very high rate observed among elderly Japanese females may be a legacy of the unusual degree of sociocultural acceptance that was traditionally accorded suicide in Japan.[26,27] Only the United Kingdom registers lower rates than the United States over most of the age spectrum. American females manifest the lowest national rates among the middle aged and elderly, and the decline in their rates after ages 45–54 is unique. Since American males do not repeat the trend, the finding probably cannot be explained by differential misclassification. However, the stigmatized nature of suicide, coupled with prohibitive clauses in life insurance policies, inevitably raises questions concerning underreporting.[6] Case ascertainment is also affected by definition. While not conclusive, a review of studies on the validity and reliability of official suicide statistics underlines a claim that these data are of sufficiently high quality to justify their use in international epidemiologic comparisons.[28] Cross-national analytic studies, which incor-

TABLE 2—Years of Life Lost (YLL) Due to Injury by Sex, Cause and Country: Age-Adjusted Rates and Percentage Distribution, 1980*

Sex/Cause	United States		France		Japan		West Germany		United Kingdom	
Male	Rate	%	Rate	%	Rate	%	Rate	%	Rate	%
Motor Vehicle	12.5	38.4	10.6	26.3	4.8	31.9	11.0	48.9	6.4	46.0
Fall	.7	2.2	.8	3.2	.7	4.8	.8	3.7	.6	4.5
Suicide	4.9	15.1	5.4	20.3	5.0	32.9	5.8	25.6	2.4	17.3
Homicide	5.8	17.9	.4	1.5	.3	2.3	.4	1.8	.4	2.9
Other Injury	8.6	26.4	9.1	48.7	4.3	28.1	4.5	20.0	4.1	29.3
All Injury	32.5	100.0	26.3	100.0	15.1	100.0	22.5	100.0	13.9	100.0
Female										
Motor Vehicle	4.1	42.8	3.2	38.4	1.2	22.9	3.4	45.6	1.7	36.6
Fall	.2	2.1	.2	2.7	.1	1.9	.2	3.2	.1	3.1
Suicide	1.4	14.4	2.0	23.5	2.3	45.0	2.2	29.4	1.1	23.5
Homicide	1.6	16.4	.2	2.6	.3	5.0	.3	4.3	.2	4.8
Other Injury	2.3	24.3	2.8	32.8	1.3	25.2	1.3	17.5	1.7	32.0
All Injury	9.6	100.0	8.4	100.0	5.2	100.0	7.4	100.0	4.8	100.0

*YLL rates per 1,000 population.

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*Personal communication, Rachid Salmi, MD, France, February 3, 1989.

porate determinants and correlates that have been identified in recent literature reviews,[29–31] may yield new insights into suicide etiology.

Although homicide accounts for fewer deaths in the United States than either motor vehicle crashes or suicide, US rates are grossly disparate from those in the comparison countries on all three measures utilized in this study. In 1980, handguns featured in an estimated 11,522 US homicides[32]; about half of the total. Handguns were also implicated in fourth-fifths of cases of firearm-related murder and nonnegligent manslaughter,[33] yet constituted only 30 percent of the national civilian firearm stock.[32] The foregoing statistics seem all the more alarming when one considers that in 1980 there were only eight handgun-related homicides in the United Kingdom,[32] which strictly regulates the sale and distribution of firearms, as do France, Japan, and West Germany. The US handgun-related homicide rate was 500 times the British rate, and the overall homicide rate was two-and-a-half times the rate of Northern Ireland,[13] a country in a state of civil war. Moreover, the homicide rate for both sexes rose by 30 percent between 1970 and 1980.[13,19] Results of a recent study suggest that restricting handgun access would be the most effective way to reduce the high US homicide rate.[34] The epidemic of firearm-related homicide (and other violence) warrants unprecedented ameliorative efforts by government agencies at all levels.

Detection of large international differences in disease rates has spawned hypotheses which, following testing, have advanced both understanding of disease etiology and prevention.[35] For example, the determination of such differences in cancer incidence rates[36,37] is credited with stimulating an array of analytic studies indicating that dietary factors have both causative and inhibitory roles in human carcinogenesis.[38] Similar progress could ensue from detection and analysis of international differentials in injury mortality and morbidity.

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4T

Determinants of Polychlorinated Biphenyls (PCBs), Polybrominated Biphenyls (PBBs), and Dichlorodiphenyl Trichloroethane (DDT) Levels in the Sera of Young Children

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Abstract: Serum samples from 285 4-year-old Michigan children were evaluated for levels of 11 environmental contaminants. Polychlorinated biphenyls (PCBs) were found in half the samples tested; polybrominated biphenyls (PBBs) in 13–21 percent; dicholorodiphenyl trichloroethane (DDT), in more than 70 percent. Nursing (Mothers' milk) was the principal source of these exposures. Congener-specific analysis documented the presence of at least one highly toxic PCB congener, 2,3',4,4',5-pentachlorobiphenyl. The data demonstrate the multigenerational impact of female exposure to persistent organic environmental contaminants. (*Am J Public Health* 1989; 79:1401–1404.)

Introduction

Halogenated polycyclic hydrocarbons are among the most ubiquitous and persistent contemporary environmental contaminants. Polychlorinated biphenyls (PCBs), once used in a broad range of industrial products, are found at particularly high concentrations in the fatty tissue of fish feeding in polluted waters, such as Lake Michigan, and in animal products from farms with contaminated silos.[1,2] Polybrominated biphenyls (PBBs), formerly used primarily as flame retardants, were accidentally mixed with cattle feed in Michigan in 1973–76, leading to widespread dairy and meat contamination throughout the state.[3] Dichlorodiphenyl trichloroethane (DDT), developed to control malaria-carrying mosquitoes, became one of the world's most extensively used pesticides in the 1950s and 1960s.[4] Dichlorodiphenyl dichloroethene (DDE) is its most stable and persistent metabolite.

PCBs, PBBs, and DDE are not readily excreted, except by lactation.[5] Placental passage has been documented for PCBs and PBBs,[6-8] although much larger quantities are transferred to the infant postnatally in breast milk due to its high lipid content.[9] A positive relation between duration of nursing and serum PCB level in young children has been reported in three studies, one in Germany[10] and two in Japan.[5,11]

Methods

The study included two cohorts of children evaluated at age 4 years. The "fish exposure cohort" consisted of 236 children recruited in 1980–81 when 8,482 women delivering infants in four western Michigan hospitals were surveyed regarding their consumption of Lake Michigan sport fish.[12] "Contaminated fish consumption" was defined as the sum of annual Lake Michigan fish consumption, weighted by the PCB content of the species consumed, in the present or past, whichever was greater.[13] All women consuming at least 11.8 kg over a six-year period (4.0 percent of those interviewed) were invited to participate in the study. A small proportion (4.6 percent) of those who did not consume these fish were also recruited and constituted 22.7 percent of the final sample. Of those assessed as infants, 75.4 percent were seen again in the present study. The children evaluated at age 4 did not differ from those not retained in the sample in terms of cord and maternal serum PCB levels but were somewhat higher in socioeconomic status (SES) and maternal fish consumption and milk PCB levels.[14]

The "farm exposure" cohort consisted of 87 children who had not previously been assessed as infants. Their families were enrolled in long-term studies of exposure to PBB or PCB contaminated farm products conducted by the Michigan Department of Public Health. The PBB families had lived on or received food from quarantined farms;[15] the PCB families had eaten milk and meat from farms whose animals consumed silage contaminated with PCBs.[16]

Information regarding family SES and residence, maternal age, weeks of breast feeding, maternal contaminated fish consumption during and before pregnancy, and child's contaminated fish consumption during the previous year was obtained by maternal interview at the four-year assessment. When compared with similar information obtained from the fish exposure cohort during infancy, test-retest reliabilities (Pearson r) ranged from 0.68 for maternal fish consumption to 0.99 for maternal age (median = 0.81).[14] The four-year maternal fish consumption report was also validated in relation to maternal serum PCB level obtained at delivery ($r = 0.37$, $P = 0.000$). Venous blood was obtained from 285 of the 323 children. Specimens were obtained from 250 children at age 4, 35 children at age 5. Levels of PCBs, PBBs, DDT, and lead did not differ significantly at the two ages.

Total serum PCB level was evaluated by the Michigan Department of Public Health by packed column electron capture gas chromatography, adapting the Webb-McCall method[17] to a computer data system, in which Aroclors 1016 and 1260 were used as reference standards.[18] Quantitation was also provided for PBBs, Total DDT (p,p'-DDT + p,p'-DDE), hexachlorobenzene, beta HCB, oxychlordane, heptachlor epoxide, trans-nonachlor, and mirex. The same laboratory had previously provided data on PCB and PBB levels in umbilical cord serum, maternal serum collected two to three days after delivery, and maternal milk for the fish

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TABLE 1—Environmental Contaminants Found in the Sera of Young Children

		Children with Trace Levels (1 ng/mL) or Greater			
	Total N	%	Mean	s.d.	Range
PCBs					
Fish exposure	205	52.2	4.18	3.29	1.00–19.40
Farm exposure	80	48.8	4.82	4.81	1.00–23.30
PBBs					
Fish exposure	205	12.7	2.44	1.50	1.00–6.40
Farm exposure	80	21.3	2.95	2.66	1.00–9.50
Total DDT					
Fish exposure	202	69.8	4.36	3.87	1.00–21.40
Farm exposure	79	77.2	4.24	4.47	1.00–143.20*
Lead†					
Fish exposure	194	—	0.30	0.16	0–1.01
Farm exposure	77	—	0.32	0.17	0.10–1.21

Means are ng/mL, except for lead μmol/L. The laboratory detection limit for PBBs and DDT is 1 ng/mL; for PCBs, 3 ng/mL. 26.3% of the fish exposure children and 23.8% of the farm exposure children had PCB levels of 3 ng/mL or greater.

*Two outliers (143.2 and 73.4) are recoded to 15 for mean and s.d. calculation, as recommended by Winer.

†Mean, s.d., and range are based on all cases tested.

exposure cohort using the same analytic method.[13] Quality control has been maintained consistently since 1979 in interlaboratory comparisons following protocols established by the Centers for Disease Control and the Environmental Protection Agency. The four-year blood samples were also analyzed for lead by atomic absorption spectrophotometry using the Delves cup method.

With the exception of the breast milk PCB and four-year blood lead levels, all of the exposure measures were positively skewed (skew > 2.5) and normalized by means of log X + 1 transformation. Means and standard deviations are reported in terms of the original distributions; the log transforms were used in the correlation and regression analyses. Child's contaminated fish consumption was measured in terms of current annual kg consumed (log X + 1) with species weighted to reflect degree of PCB contamination.[13] Weeks of nursing was transformed by recoding seven values greater than 3 standard deviations above the mean to 1 point greater than the highest observed value, as recommended by Winer.[19]

Results

PCBs were detected in about half of the 4-year-olds tested; PBBs, in relatively few samples; and DDT, though banned for many years, in a large majority of the children (Table 1). Although the mothers in the fish exposure cohort had eaten more Lake Michigan fish before and during pregnancy than their farm exposure counterparts, 7.6 kg/yr versus 1.2 kg/yr, PCB and DDT levels were similar in the two groups of children. PBBs, which had been a specific contamination problem on many of the farms, were somewhat more prevalent in the farm exposure cohort.

DDE averaged 89.0 percent of the total DDT in the children's sera, which is similar to the 86.7 percent reported for a more highly exposed, predominantly Black, rura sample in the early 1980s.[20] Except for hexachlorobenzene which was present at low levels (< 2.0 ng/mL) in three cases none of the other organochlorine pesticides tested were detected in the four-year samples. Lead levels were generally low, although 11 children had levels between 0.72 and 1.21 μmol/L.

The relation of several hypothesized determinants to four-year body burden is shown in Table 2. All predictors with zero-order correlations significant at $P < 0.10$ were also included in multiple regression analyses, which adjusted statistically for confounding among the predictors. While cord serum level was unrelated to PCB body burden at age (P = 0.615), maternal milk PCB level and weeks of breastfeeding jointly explained 59.7 percent of the variance in four-year PCB level (P = 0.000). In the absence of nursing the children's body burdens diminished with time as growth reduced the proportion of the original *in utero* dose (Table 3) Prolonged breastfeeding, by contrast, was associated with substantial increases in PCB burden. Where maternal milk PCB level exceeded 1000 ng/mL, each additional week o nursing was associated with a 0.19 ± 0.03* ng/mL increase in the child's four-year serum PCB level. Where maternal milk level was less than 600 ng/mL, the increase associated with each week of nursing was 0.09 ± 0.02 ng/mL. Socioeconomic status (SES) and maternal age were not significant predictor of four-year PCB serum level after adjustment for nursing exposure in the regression analysis. The SES effect wa attributable to the fact that higher SES mothers nursed fo longer periods; the maternal age correlation, to higher PCB levels in the milk of older mothers.

Maternal milk PBB level and weeks of nursing jointly

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*Unstandardized regression coefficient ± s.e. from a univariate regressio of four-year serum PCB level (prior to log transformation) on weeks of nursing

TABLE 2—Effects of Pre- and Postnatal Predictors on Four-year Serum Contaminant Levels

		PCBs		PBBs		DDT		Lead
	N	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted
Cord serum level	126	0.05	—	0.29	0.34	—	—	—
Maternal milk level	107	0.29	0.33	0.39	0.31	—	—	—
Weeks of breastfeeding	285	0.66	0.70	0.42	0.42	0.61	0.56	–0.11
Socioeconomic status	285	0.26	0.07	0.23	0.09	0.29	0.08	−0.21
Maternal age	285	0.15	0.01	0.09	—	0.26	0.11	−0.15
Rural residence*	285	0.01	—	0.02	—	0.12	0.13	0.03
Child's fish consumption	285	0.08	—	0.02	—	0.03	—	0.01
Multiple R		—	0.78	—	0.65	—	0.64	—

Unadjusted values are zero-order Pearson correlation coefficients. Adjusted values are standardized regression coefficients from analyses in which each contaminant was examined in relation to the set of predictors for which values are shown. All PCB, PBB, and DDT values below the laboratory's detection limits were recoded to 0 before being subjected to correlational and regression analysis.

*Coded 0 for urban or suburban, 1 for rural.

TABLE 3—Comparisons of Cord and Four-year Serum PCB Levels in Relation to Duration of Breastfeeding

Months of Breastfeeding	N	Cord Serum PCB level	Four-year Serum PCB level
0.0–2.9	66	2.8	0.6
3.0–5.9	17	2.2	2.3
6.0–11.9	30	2.6	4.4
12.0⁺	13	2.1	5.5

Means are ng/mL.

explained 31.5 percent of the variance in four-year serum PBB level (P = 0.000). Although four-year PBB body burden was correlated with cord serum level as well, inspection of a scatterplot relating the breast milk and four-year PBB levels suggested that the relation between these variables was curvilinear. When a quadratic term for milk level was added to the regression, the multiple R increased significantly to 0.69, and the standardized regression coefficient for cord serum PBB level was no longer significant, β = 0.10, P = 0.384. Moreover, a case by case examination of the data on which the PBB regression was based indicated that all the children with detectable four-year serum PBB levels had been nursed for at least four months.

Although no data were available on breast milk DDT levels, duration of nursing explained 36.8 percent of the variance in four-year serum DDT level (P = 0.000). DDT levels were also higher in children in rural areas and in the children of older mothers, even after adjustment for weeks of nursing (Ps = 0.007 and 0.032, respectively). Because the total DDT in these children consisted primarily of DDE, the correlation and regression coefficients for these predictors in relation to DDE were virtually identical to those shown for total DDT. No regression was computed for lead because the magnitude of its relations with these predictors was small.

The Webb-McCall isomer identification method records chromatographic elution peaks, each of which is comprised of one or more specific PCB congeners (Figure 1). Peaks 203, 232/244, 332, and 372 were statistically more prevalent in the farm exposure cohort, suggesting that the farm children may have been exposed to sources of PCBs to which the fish cohort children were not exposed. Despite these differences, the overall pattern of elution peaks was highly similar in the two cohorts and closely matched the pattern found by Humphrey[21] in adults and regular fish eaters. Although many different PCB mixtures have been deposited in the environment,[22] these data confirm that certain congeners are preferentially accumulated in both adults and children.

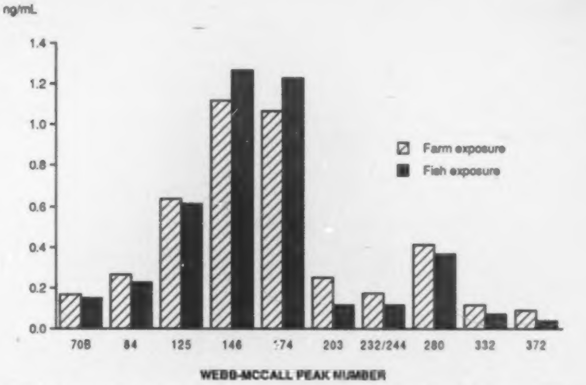

FIGURE 1—Webb-McCall chromatographic elution peak values by exposure cohort.

Serum extracts from 20 children with PCB serum levels in excess of 3.0 ng/mL were pooled into two composite samples—one for the boys (N = 9); the other for the girls (N = 11)—and analyzed at the US Environmental Protection Agency Large Lakes Research Station at Grosse Ile, Michigan, by means of capillary column gas chromatography.[23] The type and concentration of PCB congeners found in the two pooled samples were highly consistent (Table 4), making it unlikely that the pattern observed was determined by one or two aberrant children. The most prevalent congeners, Nos. 153, 138, 180, and 118, were also among the most prevalent in human milk and serum samples evaluated in prior studies.[25–29] No. 118, a constituent of Webb-McCall Peak 125 (Figure 1), is a mono-*ortho* analog of the highly toxic

TABLE 4—Most Prevalent Congeners in Pooled Serum Samples at Age 4 Years

Congener No.[24]	Structure	Sample 1 (11 females) Mean	Sample 1 (11 females) %	Sample 2 (9 males) Mean	Sample 2 (9 males) %
153	2,2',4,4',5,5'	1.50	19.6	1.30	15.8
138	2,2',3,4,4',5' *	1.30	17.0	1.15	14.0
163	2,3,3',4',5,6				
180	2,2',3,4,4',5,5'	0.62	8.1	0.56	6.8
118	2,3',4,4',5	0.35	4.6	0.53	6.5
170	2,2',3,3',4,4',5	0.35	4.6	0.32	3.9
005	2,3 *	0.27	3.5	0.04	0.5
008	2,4'				
099	2,2',4,4',5	0.22	2.9	0.22	2.7
187	2,2',3,4',5,5',6 †	0.21	2.7	0.16	2.0
182	2,2',3,4,4',5,6'				
031	2,4',5 *	0.21	2.7	0.22	2.7
028	2,4,4'				
018	2,2',5	0.14	1.8	0.15	1.8
Total		7.65‡	67.5§	8.20‡	56.7§

From capillary column gas chromatographic analyses. Means are ng/mL.
*Sum.
†Average.
‡Total PCBs from the pooled sample.
§Percentage of the sample comprised of the congeners listed on this table.

coplanar PCB congeners, which elicit effects resembling those caused by 2,3,7,8-TCDD. This congener, which has also been detected at relatively high concentrations in human milk in the US,[25,26] Germany,[27] and Japan,[28] is likely to provide a major contribution to the overall toxicity of these children's PCB exposure.[26,30]

Discussion

The present data implicate nursing as the principal source of PCB and PBB exposure in these children and a significant source of DDT exposure as well. Although prenatal PCB exposure has been shown to affect physical and behavioral development,[12,31,32] the quantity transferred *in utero* is apparently too small to detect in serum by age 4 years. In contrast to transplacental exposure, developmental deficits have not been linked to postnatal nursing exposure in North American children,[33,34] although dermatological effects from nursing have been documented in the infants of occupationally exposed women in Japan.[35]

Postnatal exposure to the halogenated hydrocarbon contaminants was greater for higher SES children, because their mothers tended to breastfeed for longer periods. Postnatal fish consumption by the child had little effect on PCB body burden, because most children ate less than one fish meal per year. While virtually all of the children with PCB levels of 3.0 ng/mL or greater had breastfed for at least six weeks, one child who had nursed for only two weeks but had eaten 84 contaminated fish meals in the previous year also had an elevated PCB body burden at age 4.

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2T

Commentary

Fluoridation Referendum in La Crosse, Wisconsin: Contributing Factors to Success

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Abstract: Residents of La Crosse, Wisconsin approved a public referendum in favor of water fluoridation on April 5, 1988. The vote, 57 percent supportive, culminated a two-year community effort. Three public referenda had been defeated in the past. Contributing to the success of this recent campaign were: broad-based community support led by a 34-member Citizens for Better Dental Health in La Crosse Committee; American Dental Association/Wisconsin Division of Health/US Public Health Service consultation and support; knowledgeable and supportive press coverage; the timing of the ballot to coincide with the Wisconsin Presidential Primary; and local chiropractic support to offset chiropractic anti-fluoridation leadership. La Crosse, population 50,000, was the largest fluoride-deficient community in a nine-state upper Midwest area. (*Am J Public Health* 1989; 79:1405–1408.)

Introduction

Water fluoridation, the purposeful supplementation of naturally occurring fluoride to optimal levels for the prevention of dental caries, is one of the most successful public health measures in history.[1-3] Nevertheless, despite over 40 years documentation of safety, efficacy, cost-effectiveness, and practicality, and the unqualified endorsements of every major health organization, water fluoridation is far from universal in the United States. At the present time, approximately 63 percent of the US citizens have access to community drinking water containing dentally significant levels of fluoride (0.7 ppm).*

From a public health perspective, centralized decision-making by local health administrations affords the best opportunity for fluoridation implementation in the 42 states which have no legislatively mandated fluoridation.[4,5] Unfortunately, decisions on fluoridation in many communities are made in a highly charged political arena.[6,7] A number of fluoride-deficient communities in the US have failed to fluoridate because of initiative defeat in public referenda.[8,9] Fluoridation remains one of the few, if not the only, public health measures to be put to the vote of the public. Of the 150 referenda recorded by the Dental Disease Prevention Activity, Centers for Disease Control from 1980 through 1988, 64 percent (96) ended in failure for fluoridation proponents. Oftentimes proponents are ill-prepared for the political nature of a fluoridation campaign. By contrast, over the same time period, in 258 communities where the decision was left to administration (city councils or health boards), 77 percent (199) implemented fluoridation* (Table 1).

The purpose of this discussion is to outline key contributing factors in a recently successful fluoridation initiative in La Crosse, Wisconsin. A 20-month community effort resulted in a 57 percent positive vote for fluoridation. These contributing factors to success may be applicable to fluoridation strategies in other fluoride-deficient communities where the issue is decided by public referendum.

La Crosse Historical Perspective

La Crosse, 50,000 population, was the largest fluoride-deficient community (0.1 ppm) in a nine-state upper Midwest region. Although 93 percent of Wisconsin residents on public water supplies have optimally fluoridated water, La Crosse has a long history of antifluoridation obstructionism. On three occasions, voters rejected fluoridation in public referenda despite widespread support from health professional organizations. In 1953, by council action, the city fluoridated for three months until antifluoridation forces led by area chiropractors pressured officials to reconsider. Soon after, a first referendum failed with only 18 percent positive vote. The fluoridation equipment was disconnected and sold.

A second referendum failed in 1967 with 43 percent support; a third in 1969 gained only 29 percent support. Antifluoridation influence continued in the 1970s and 1980s driven by a prominent local chiropractor whose family had led the fight against fluoridation for over 30 years. Tactics used by La Crosse anti-fluoridationists through the years copied national anti-strategies capitalizing on the concerns of

TABLE 1—Fluoridation Implementation in US Communities, by Decision-making Process, 1980–88

	By Referendum		By Governing Body	
Year	Yes	No	Yes	No
Year				
1980	7	33	19	14
1981	4	10	26	8
1982	6	13	28	6
1983	6	13	20	14
1984	6	5	37	4
1985	5	4	15	4
1986	6	9	21	4
1987	4	3	18	5
1988	10	6	15	0
Total	54 (36%)	96 (64%)	199 (77%)	59 (23%)

SOURCE: Dental Disease Prevention Activity, Centers for Disease Control.

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*Dental Disease Prevention Activity, Centers for Disease Control, Atlanta, GA.

society at the time. Fluoridation was alleged to be a Communist plot in the 1950s, a cancer causing "toxic waste" and "poison" in the 1960s and 1970s, and by the 1980s linked to aging, Alzheimer's disease, AIDS and "freedom of choice."

Renewed Fluoridation Initiative

La Crosse community leaders renewed fluoridation efforts in 1986 in response to a perception that a majority of a new city council would be supportive. A local committee was formed led by a representative of the La Crosse District Dental Society and the county public health department director. Efforts were directed toward convincing an 18-member city council to implement fluoridation without public referendum by convincing councillors of the health benefits of fluoridation as well as demonstrating public support for the initiative. Community support was strong through four public hearings and led to a June 1987 city council 10 to 8 vote for fluoridation, the first positive vote in 34 years. Despite the positive vote, the council delayed funding for 60 days thus allowing opponents the opportunity to petition for public referendum. This allowed fluoridation opponents to obtain the 3,000 signatures necessary to force a referendum. The pro-fluoridation majority of the council set the date for the referendum at the next major election, April 5, 1988, the Wisconsin Presidential Primary, rather than at an earlier special election date for a school bond. The wording of the ballot, an important component of successful fluoridation referenda, read in a straightforward and simple manner as follows: "Should the City of La Crosse fluoridate its water supply?"

Factors Contributing to Success

When compared to the previous defeats, the following five factors could be identified:

Broad-Based Community Support

A broad support base is necessary to assure success once a fluoridation issue is politicized.[4,8] Soon after the establishment of the referendum date, the original pro-fluoridation committee formed a campaign organization, "Citizens for Better Dental Health in La Crosse." The organization included representatives from health professional associations, the public health sector, hospitals, business and industry, labor, university, and civic and community organizations. The Citizens for Better Dental Health met twice a month for five months preceding the April referendum, planning and implementing an educational and political campaign for fluoridation. A subcommittee structure centered on a number of campaign tasks. The agenda reflected a strong local commitment with knowledgeable and credible spokespersons. Strategy for political success included: no debates or public forums that give antifluoridationists credibility; no distractions from a central theme of better health; less time on technical merits and more time on targeting groups of voters, identifying supporters and encouraging their turnout (see Appendix for further details).

Scientific Press Coverage

Scientific evaluation of the fluoridation issues by an influential press was an asset to the fluoridation initiative. Print media coverage in La Crosse was extensive and well-researched. The *La Crosse Tribune*, with a circulation of 40,000, is the largest newspaper in southwest Wisconsin. The fluoridation issue provoked much public debate as evidenced by 142 letters-to-the-editor (pro and anti) published by the newspaper during the course of the initiative.

It was of great benefit that the health reporter for the *Tribune* (a college chemistry major) was knowledgeable in the history, science, and efficacy of water fluoridation. This resulted in positive and informative health reporting on the issue. Headlines reflected positive leads such as "Fluoride—History Shows No Reason for Controversy—What's the Fuss?" Supportive editorials as one entitled "Let's Get Beyond the Pseudo-Science" appeared throughout the campaign. When spurious newspaper advertisements were run by opponents, follow-up stories refuted erroneous claims. For example, an antifluoridation paid advertisement was entitled "Fluoridation Causes Corrosion of Pipes. Vote No to Fluoridation." The following day an article appeared discrediting the corrosion charge through interviews with the La Crosse Water Utility manager and Centers for Disease Control officials in Atlanta, GA and citing research and fluoridation endorsements from the National Institutes of Health, Bell Laboratories, the American Water Works Association, and the National Association of Corrosion Engineers. The decision of the local press to forego sensationalizing the issue in deference to accurately educating the public was a critical factor toward success.

ADA and Governmental Agency Support

The La Crosse committee utilized a number of sources for consultative support. The American Dental Association (ADA) provided technical and financial support. National experts gave council testimony and committee consultation through ADA sponsorship. The ADA's Water Fluoridation Campaign Package and other written and audio/visual materials were provided. ADA letters of endorsement were sent to the city and the *La Crosse Tribune*.

The Wisconsin Division of Health through the State Dental Director's office provided technical assistance throughout the course of the initiative. Literature reviews, strategy development, and analyses of other fluoridation referenda were utilized by the committee. A state cost-sharing grant for fluoridation equipment was available to the community.

The community also used the services of the US Public Health Service. Consultation and letters of support were utilized through the Dental Disease Prevention Activity of Centers for Disease Control in Atlanta and the US Surgeon General's Office, Washington, DC. The most notable letter of support was from US Surgeon General C. Everett Koop. Dr. Koop's letter to the *La Crosse Tribune*, published just prior to the referendum date, applauded the fluoridation initiative. It was accompanied by a front page article entitled "Surgeon General to Voters: Fluoridate."

Wisconsin Presidential Primary

If fluoridation approval is subject to public referendum, the timing of the ballot is an important factor for success. It is generally perceived that fluoridation referenda should coincide with primary or general elections that promise the largest voter turnout ensuring a broader community participation rather than in smaller special issues elections.[8] The La Crosse referendum was set to coincide with the 1988 Wisconsin Presidential Primary. Fortunately, the Wisconsin Primary in the spring of 1988 was of national significance. Presidential candidates George Bush, Michael Dukakis, Jesse Jackson, and Albert Gore all campaigned in the La Crosse area. Voter interest and turnout was high. A pre-election *La Crosse Tribune*-sponsored poll was conducted by the political science department of the University of Wisconsin-La Crosse found 58 percent of city voters in favor of fluoridation, 27 percent opposed, and 15 percent undecided (Table 2). Support for fluoridation was highest among the better educated and younger voters. Among college graduates, 71 percent favored fluoridation while 62 percent of those with some college and 37 percent of those who never attended college supported the issue. Only 41 percent of citizens over age 65 supported fluoridation. Post-election analysis of voter turnout identified high participation of younger voters who had been targeted by the profluoridation organization.

Chiropractic Support

Chiropractors were among early opponents to fluoridation in the United States. Some continue to oppose fluoridation in many areas as being an infringement of personal freedom, a dictate of organized medicine, and incompatible with chiropractic nutritional philosophy.[10,11] More recently, some chiropractors are active as proponents to fluoridation and several chiropractic organizations are reaching out to the scientific community by endorsing the scientific principles of public health.[12]

For the first time in La Crosse, chiropractors were on both sides of the fluoridation issue. A few chiropractors presented supportive public testimony and actively campaigned for fluoridation. The controversy in the local chiropractic community gained statewide attention, often overshadowing other issues, and may have contributed to a loss of credibility for the primary chiropractic opponents to fluoridation. Sentiments of a proponent chiropractor are reflected in this statement which appeared in a Madison, Wisconsin newspaper: "Chiropractors are interested in public health. Fluoridation is an extremely important issue. Chiropractors have a drugless healing profession, but the feeling that fluoridation is medication is absurd. It is an essential nutrient that naturally occurs."[13]

Summary

The majority of public referenda on water fluoridation in US communities are defeated at the polls. A "confusion hypothesis" has been formulated to explain such a continuing negative voter response to fluoridation initiatives.[7,10,14] Voters are often confused by apparent contradictory information from proponents and opponents of fluoridation. The general public, unable to evaluate highly technical information, credits antifluoridationists as scientific spokespersons. The scientific community has not been all that effective in helping the public understand who is an expert and who is not.[7]

In La Crosse, the Citizens for Better Dental Health were able to overcome a long history of antifluoridation influences through educational and political strategies. Broad-based community support, consultative assistance, scientific press coverage, the timing of the ballot, and chiropractic support all contributed to the success of the initiative.

TABLE 2—Fluoridation Support by Age Group: Pre-Referendum Survey

Age	% Favor	% Oppose	% Undecided
18–25	61	12	27
26–34	66	24	10
35–54	62	28	10
55–64	58	25	17
Over 65	41	48	11
Overall	58	27	15

SOURCE: La Crosse Tribune, March 27, 1988

APPENDIX A
La Crosse Fluoridation Campaign Activities

Citizens for Better Dental Health in La Crosse

Committee with broad-based community coalition.

Endorsement by physicians and dentists.

Endorsement by civic and health organizations (American Cancer Society, local hospitals, Committee on Aging, Chamber of Commerce, AFL/CIO).

Campaign Headquarters

Placed in a highly visible downtown center location.

Opened headquarters with a press conference.

Coordinated campaign activities.

Created headquarters' theme: "Come in and take the taste test" (to counter an anti misinformation campaign regarding the taste of fluoridated water).

Speakers' Bureau

Provided knowledgeable public speakers in support of fluoridation.

Assigned speakers to civic organizations, labor groups, parent associations, and others.

Media Strategies

Identified media contact expert interviewees.

Organized positive and timely letters-to-the-editor.

Developed newspaper and radio advertisements.

Campaign Literature

Designed informational brochures for direct mail distribution, door-to-door literature drop, speaking engagements, and community locations (malls, businesses, professional offices).

Printed and distributed yard and window signs for homes, schools, and businesses.

Fund Raising

Solicited $10,000 in donations from civic/professional organizations, foundations, business, and individuals.

Print design and media production donated by a major local industry.

Getting Out the Vote

Identified and registered fluoridation supporters.

Organized special initiatives i.e., schools (parent-to-parent contact) and university (student voter registration drive).

Arranged car-pooling assistance to the polls.

Assisted with absentee ballots.

Utilized 750 volunteers in the campaign.

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Alfred Yankauer, MD, MPH, to Retire as Journal Editor; Search Committee Actively Seeking New Editor

Alfred Yankauer, MD, MPH, has indicated he plans to retire as editor of the *American Journal of Public Health* next year, effective July 1 1990. The announcement of his retirement appeared in the September issue of *The Nation's Health*, along with information about the newly appointed search committee—which is soliciting nominations for his replacement—and a list of the qualifications for the position of editor.

Dr. Yankauer will complete his 15th year as editor before stepping down next summer; his service spans the period July 1975–July 1990. Only two editors have served longer: George Rosen, MD, who preceded Dr. Yankauer, served for 16 1/2 years (August 1957–December 1973), and Mazÿck P. Ravenel, MD, served 16 years as editor (January 1925–December 1940) and was then named emeritus editor in 1941. All three editors were members of the editorial board or the editorial committee before being named editor. Fourth in line for length of service was C.-E. A. Winslow, DrPH, who held the position for 10 1/2 years (April 1944–November 1954) before retiring for health reasons.

Since the *American Journal of Public Health* was established in 1911, only 11 persons have occupied the position of editor. The four editors named above filled the role collectively for 58 years, while seven others shared the honor a total of 22 years.

In announcing his desire to retire, Dr. Yankauer stated that, "It was a decision not lightly made," because he said he has both enjoyed and learned from the experience. "However, at this point in my life, there are other things that I want and need to do," he added.

Each of the 11 editors of the Journal served that post with distinction and made a lasting impact on the Journal, its readers, the public health profession, and the health of the public. Under their respective leadership over eight decades, the Journal has grown in stature and scientific credibility. Its reputation for accuracy, relevance, and timeliness in the field of peer-reviewed professional journals has been maintained and enhanced by their devotion to its content and to its objectives.

Over the years, the Journal's table of contents has reflected current thinking about the health status of mankind and what needs to be done to improve it. The Journal has been a repository throughout the 20th century for articles, studies, and evaluations authored by hundreds of dedicated health professionals.

As the Editor Search Committee carries out its difficult task of identifying potential candidates for the position of Journal editor, they will be seeking an individual with broad public health interests, a strong historical perspective, an abiding concern for the health and well-being of others, and exceptional vision.

Nominations for Journal editor should be submitted as soon as possible, so that the search committee can complete its deliberations by the end of this year. Names of potential candidates, along with support materials, should be sent to the Editor Search Committee, c/o Executive Director's Office, American Public Health Association, 1015 15th Street, NW, Washington, DC 20005. A list of the criteria for selection of an editor appeared in the September issue of *The Nation's Health*, or may be obtained on request to APHA headquarters. Tel: (202)789-5600.

Public Health Briefs

Cocaine and Heroin Dependence Compared: Evidence from an Epidemiologic Field Survey

JAMES C. ANTHONY, PHD, AND KENNETH R. PETRONIS, MS

Abstract: Analyzing self-report interview data from a multi-site collaborative study of mental disorders in the community, we examined suspected differences between 611 cocaine users and 126 heroin users in relation to indicators of drug dependence. Cocaine users were less likely to report dependence-related problems as compared to heroin users. These epidemiologic sample data underscore concerns about the generalizability of published clinical sample data on self-reported cocaine dependence. (*Am J Public Health* 1989; 79:1409–1410.)

Introduction

Recently Hasin, *et al*, described 75 cocaine users and 67 heroin users, drawn from 308 patients in an alcohol treatment unit. Answering standardized drug questions in the Diagnostic Interview Schedule (DIS), cocaine users reported having felt dependent on cocaine just as frequently as heroin users reported having felt dependent on heroin. Further, cocaine users and heroin users differed little on other reported dependence indicators (withdrawal, loss of control, tolerance). While citing this as evidence that cocaine has dependence-generating potential comparable to that of heroin, the authors cautioned that drug users in treatment might have had different experiences than users in the general population.[1]

Previously, our research group reported on cocaine use and cocaine problems in the community, based on DIS data from the National Institute of Mental Health (NIMH) Epidemiologic Catchment Area (ECA) Program.[2] For this paper we re-analyzed the data for direct comparison to the data reported by Hasin.[1]

Methods

Collaborators at four ECA sites obtained interviews with 13,538 subjects between 1981 and 1984 after drawing probability samples of adult household residents in Baltimore, Maryland, St. Louis, Missouri, Durham, North Carolina, and Los Angeles, California. To assess psychiatric conditions, including drug-related problems, staff administered the DIS soon after sampling. The mean participation rate was 76 percent (range 68–79 percent). (New Haven, Connecticut ECA data sets do not include drug variables needed for this analysis.)

Hasin, *et al*, describe psychometric characteristics of the DIS.[1] Elsewhere, we have published data from a DIS validity study,[3] descriptions of DIS drug data,[2,4,5] and reports on sampling and other methods used in the ECA program.[6,7]

Analyses for this paper follow specifications of the clinical sample paper. The results shown are unweighted characteristics of the sample; use of sampling weights increases differences between the two studies.

Results

Among ECA subjects, 57 reported having used heroin every day for at least two weeks ("daily heroin use"); 69 reported using heroin at least six times, but never daily for two weeks. An additional 611 subjects reported having used cocaine (but not heroin) at least six times; 69 of these users reported having used cocaine every day for at least two weeks ("daily cocaine use"). These users were sociodemographically similar to those in the clinical sample (data available upon request to authors). Whereas alcohol disorders affected virtually all of the clinical sample, in the ECA sample they affected only 35 percent of the non-daily cocaine users, 52 percent of the daily cocaine users, and about 60 percent of the heroin users.

Table 1 shows for daily and non-daily cocaine and heroin users in the ECA sample the proportion who reported feeling dependent on cocaine, unsuccessfully cutting down on cocaine use, cocaine tolerance, or cocaine withdrawal.

ECA cocaine users were less likely to report cocaine-related problems than users in Hasin's study: 2.6 percent of ECA non-daily users and 22.1 percent of ECA daily users reported feeling dependent on cocaine compared to 14 percent and 79 percent, respectively, in Hasin's study; for unsuccessfully attempting to cut down on cocaine use, corresponding figures were 2.4 percent and 11.6 percent vs 14 percent and 64 percent; for tolerance, 7.7 percent and 42.0 percent vs 26 percent and 64 percent. Finally, 1.0 percent of ECA non-daily users and 13.0 percent of daily users reported feeling withdrawal sickness upon stopping or cutting down on cocaine use, as compared to 19 percent and 76 percent in Hasin's sample.

Our sample and Hasin's sample also differed in relation to heroin experience, although not as markedly as with cocaine. Among ECA non-daily and daily heroin users, respectively, 7.2 percent and 78.6 percent reported having felt dependent on heroin, as compared to 11 percent and 85 percent in Hasin's study. For loss of control, comparable figures were 1.5 percent and 40 percent vs 16 percent and 63 percent; for heroin tolerance, 12 percent and 68.4 percent vs

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TABLE 1—Dependence Indicators for Cocaine and Heroin, by Patterns of Use, based on Data from the Epidemiologic Catchment Area Surveys in Baltimore, St. Louis, Durham, and Los Angeles, 1980–84

	Cocaine				Heroin			
	Group 1 (N = 542 6+ times, Never daily for 2 weeks)		Group 2 (N = 69 Daily 2 weeks or longer)		Group 3 (N = 69 6+ times, Never daily for 2 weeks)		Group 4 (N = 57 Daily 2 weeks or longer)	
Dependence Indicators	%	n	%	n	%	n	%	n
Ever felt dependent on drug	2.6 (4 missing)	14	22.1 (1 missing)	15	7.2	5	78.6 (1 missing)	44
Ever unable to cut down on amount used	2.4	13	11.6	8	1.5	1	40.4	23
Ever felt tolerance to drug's effects	7.7 (7 missing)	41	42.0	[illegible]	11.8 (1 missing)	8	68.4	39
Ever experienced withdrawal	0.9 (3 missing)	5	13.0	9	10.1	7	75.4	43

Differences in proportions between groups 1 vs 3 and groups 2 vs 4 were as follows (95% confidence limits):

	Group 1 vs. Group 3	Group 2 vs. Group 4
Felt dependent	−4.6 (−10.9, 1.6)	−56.5 (−71.1, −41.9)
Unable to cut down	0.9 (−2.2, 4.1)	−28.8 (−43.6, −14.0)
Tolerance	−4.1 (−12.1, 3.9)	−26.4 (−43.2, −9.6)
Withdrawal	−9.2 (−16.4, −2.1)	−62.4 (−76.1, −48.7)

26 percent and 85 percent; for heroin withdrawal experience, 10 percent and 75 percent vs 21 percent and 83 percent.

Unlike the cases reported by Hasin, ECA daily cocaine users were much less likely than daily heroin users to report problems, as shown by negative signs on the upper confidence limits for each estimated difference (Table 1).

Discussion

This epidemiologic study's evidence calls into question the generalizability of results from the prior study of patients in an alcohol treatment unit. Responding to identical self-report interview questions, smaller proportions of cocaine users in the community sample reported dependence-related problems. Moreover, daily cocaine users in this sample were substantially less likely to report dependence-related problems, as compared to daily heroin users.

One hypothesis about these differences in study results is that drug users in alcohol treatment are more willing to give a complete report about drug-related problems. Alternatively, alcoholism or admission to alcoholism treatment may index especially intensive patterns of drug use or increased risk for problems with other drugs, especially cocaine. If so, clinical samples are unreliable sources of data on the frequency of drug hazards in the community, as discussed elsewhere.[1,2]

Putting these results in perspective, there is strong laboratory evidence on reinforcing properties of cocaine. Across several species, cocaine is self-administered repetitively in patterns that fulfill broadly accepted case definitions for drug dependence.[8,9] These interview data do not contradict that evidence.

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Improving Patient Comprehension of Literature on Smoking

Cathy D. Meade, RN, PhD, James C. Byrd, MD, MPH, and Martha Lee, PhD

Abstract: This study was designed to determine if simplification of smoking literature improved patient comprehension. Subjects (N = 129) were randomized in three groups: to receive no smoking booklet, receive one written at the 5th grade, or the 10th grade reading level. Subjects receiving the 5th grade booklet showed 13 percent better comprehension than those receiving the 10th grade booklet, and 18 percent better than the controls. Comprehension of written smoking materials can be improved by adjustment of the reading grade level. (*Am J Public Health* 1989; 79:1411–1412.)

Introduction

Printed materials on smoking are commonly distributed to patients in hospitals, clinics, or smoking cessation programs.[1–5] Such materials communicate, reinforce, and extend the educational message presented verbally by practitioners.[6,7]

Studies indicate that the median level of most patient educational materials is grade 10, a reading level often greater than the majority of its intended target population.[8–13] In a review of 49 educational brochures specific to smoking, the median reading level was 10.5 while the median reading level of patients (N = 258) was grade 6.[14]

Comprehension and acceptability of written materials should improve if materials are made easier to read; however, few data are available to either support or refute this idea. Reported studies have lacked control groups, pretest measures,[15–17] randomization, or description of reliability of the instruments.[15–16] No study reported has yet evaluated the effect of simplification of smoking literature on patient comprehension. We report here such a study.

Methods

Two booklets on smoking, each entitled "Quit Smoking and Feel Good," were developed based on a review of the smoking literature and a panel of experts. The booklets mirrored each other in color, design, writing style, print type, length, and organization. The Minnesota Interactive Readability Approximation Microcomputer Program was used to adjust the readability level of one booklet to grade 5 and the other to grade 10.[18]

To evaluate smoking knowledge and recall, 24 questions were developed by the investigators with a panel of experts based upon the content of the booklets. The reliability of the test was found to be at an acceptable level (r = 0.73 Cronbachs alpha).[19] Questions were systematically divided into two 12-question tests (pretest and posttest) based on results of the reliability measures, item difficulty, logical pairing of questions, and question content. The pretest and posttest were each comprised of six multiple choice and six true/false questions.

Smoking subjects (N = 129) were selected from three Primary Care clinics based in a public hospital, a veterans hospital, and a community health center. Selection criteria included being 18 years or older, English speakers, English readers, visually able to read 12 point typeset, and able to give free consent.

Subjects were randomly placed into one of three groups: control, experimental group 5 (E5), or experimental group 10 (E10). All subjects were first administered the pretest. Control group subjects (N = 44) received no smoking booklet. Subjects in E5 (N = 44) received a smoking booklet written at grade 5 reading level, those in E10 (N = 41) one written at a grade 10 reading level.

All subjects were administered the posttest, the Wide-Range Achievement Test (WRAT) Level II (a word recognition test which estimates reading grade level)[20] and were then asked questions about their educational background and other demographic information.

Results

The sample was comprised of 74 males (57 percent) and 55 females (43 percent); ages ranged from 18 to 88 with a mean of 43 years. There were 85 Blacks (66 percent) and 44 Whites (34 percent). Chi-square analyses revealed that there were no significant differences in sex, ethnic background, income, education, or WRAT between control, E5, or E10. Age was significantly different between the groups, with a mean of 49 years for control, 41 years for E5, and 40 years for E10. The mean grade level of reported education among subjects was grade 11 and the mean grade level according to the WRAT was grade 7, a difference of 4 grades (paired t = 12.62, 95% CI: 3.31, 4.52).

Prescores, postscores, and score differences were calculated for the three groups (Table 1). The score improvement (Postscore − Prescore) ÷ Prescore, showed a 4 percent increase in scores for the control group, a 22 percent increase for E5, and a 9 percent increase for E10. Using one-way analysis of variance, no significant differences were found in the prescores between control, E5, or E10. Significant

TABLE 1—Summary of Scores by Booklet Group

	Booklet Groups		
Range	Control (N = 44)	E5 (N = 44)	E10 (N = 41)
Prescore (3,12)			
Mean	6.68	7.25	7.17
SD	2.42	2.16	2.24
Postscore (1,12)			
Mean	6.93	8.82	7.80
SD	2.50	2.33	2.84
Score Difference (−6,6)			
Mean	0.25	1.57	0.63
SD	2.28	2.54	2.38
95% CI	(−0.44,0.94)	(0.80,2.34)	(−0.12,1.39)
Score Improvement (Postscore − Prescore) ÷ Prescore	3.7%	21.6%	8.7%

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TABLE 2—Comparison of Scores by Booklet and WRAT

Groups	Booklet*/ WRAT	N	Prescore (Mean ± SD)	Postscore (Mean ± SD)	Score Difference	95% CI
I	Low/low	23	6.43 ± 1.75	7.87 ± 2.69	1.43	(0.21,2.66)
II	Low/high	21	8.14 ± 2.20	9.86 ± 1.24	1.71	(0.70,2.73)
III	High/low	20	5.90 ± 2.00	6.05 ± 2.86	0.15	(−1.11,1.41)
IV	High/high	21	8.38 ± 1.77	9.48 ± 1.54	1.10	(0.19,2.00)

*Low booklet = 5th grade reading level booklet
High booklet = 10th grade reading level booklet
High WRAT = WRAT score > grade 7
Low WRAT = ≤ grade 7

differences were found between the groups in their postscores ($p = 0.0033$), and in their raw score differences ($p = 0.0335$).

Subjects in groups E5 and E10 (N = 85) were subdivided into four groups according to their WRAT score and grade level of booklet received (Table 2). Definitions for high and low booklet and high and low reader were based on expected reading proficiency of subjects within a two year range of the readability of the available study booklets. A comparison of scores was then conducted to investigate differences between the groups.

Results indicated that subjects with high WRAT scores had higher prescores and postscores than subjects with low WRAT scores. The largest score differences (1.43 and 1.71) were noted in the groups (I and II) who received the fifth grade booklet regardless of their WRAT scores. The group which exhibited the smallest increase in score difference (0.15) was the low reader who received the tenth grade booklet (Group III).

Using the prescore and WRAT as covariates, results indicated that the booklet had significant main effects on score differences ($p = 0.0002$). Adjusted score difference means for control was 0.08 (SEM = 0.3028), for E5 was 1.73 (SEM = 0.3005), and for E10 was 0.81 (SEM 0.3115).

Discussion

Since education has been noted as a factor in smoking cessation programs, success may be related to the participants' acceptability and understandability of the information.[1] Our results support the need for continued development of educational materials at low reading levels. Producers of literature on smoking need to apply reading analyses to their texts. Readability formulas can assist in estimating the reading level of materials by offering an objective measure of text difficulty. Factors such as format, visual appeal, patient interest, and motivation also need to be considered when writing health messages.

We suggest that user tests (pretests and posttests) be incorporated into smoking cessation programs to help determine if a message is being comprehended by the participant. Such tests can offer information on clients' smoking knowledge over time when planned interventions are administered.[21–23]

Findings also indicated that educational level is a poor predictor of reading ability. Years of schooling do not identify reading ability, a finding supported by other investigators.[9,11,14,24] We suggest that an instrument such as the WRAT be used as a more accurate predictor of reading skills. The WRAT can be administered and scored in a manner of minutes.

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17

Cigarette and Smokeless Tobacco Use among Connecticut Adolescents

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Abstract: Despite data that the rate of boys' cigarette use has declined nationwide, boys' total tobacco use may actually have remained high because of their substantial use of smokeless tobacco. We surveyed anonymously a random sample of entire classes of grade 7–12 boys and girls from 59 Connecticut towns concerning tobacco use. Despite girls' higher daily cigarette use, boys' total daily tobacco use was higher than that of girls in grades 7–9, and only slightly lower in grades 10–12. (*Am J Public Health* 1989; 79:1413–1414.)

Introduction

Boys' cigarette use has generally been perceived as having peaked in the late 1960s (14.7 percent regular use in 1968 across grades 7–12), declined by 1979, and stabilized[1] with girls smoking more than boys. In a 1979 survey of grades 7–12, girls reported regular smoking at a rate of 12.7 percent and boys 10.7 percent.[1] The annual National Institute on Drug Abuse (NIDA) survey of high school seniors indicates a daily smoking rate of 18.7 percent in both 1986 and 1987 and a monthly rate of 29.4 percent in 1987, consistent since 1979.[2]

The mid-1970s might have marked the beginning of a large increase in adolescent smokeless tobacco use, first recognized in print a few years later.[3,4] By the time of the Surgeon General's 1986 report on smokeless tobacco,[5] it was apparent that many boys were using smokeless tobacco in grade school and continuing its use throughout high school. Minimal smokeless tobacco use by females has been reported except in particular regional pockets.[6–11] Since many effects of the two products are identical, it seems appropriate to document total tobacco use for both girls and boys.

As part of our research on smokeless tobacco use, we conducted a 1987 statewide survey in Connecticut assessing total tobacco use in grades 7–12. Based on surveys,[5,12] and on earlier data from Massachusetts,* we expected to find relatively low smokeless tobacco use.

Methods

Subjects and Design

All 169 Connecticut towns were blocked by population density and socioeconomic status (SES). Within the resultant matrix, proportional numbers of towns were chosen randomly, and middle and high schools were selected randomly from these towns. Requests were made sequentially to public school superintendents (75 percent agreed) and then to principals (89 percent agreed) for permission to collect anonymous, self-administered student questionnaires. Schools whose superintendents or principals refused permission were replaced with schools of similar population density and SES. Schools were asked to choose entire classrooms of students such that either regular or a combination of high and low academic track students would be represented. Further blocking by sex and grade within school was carried out in order to collect approximately 3 percent of all Connecticut students in grades 7–12 representing public, town-based schools.

Questionnaires, which were sent to and returned from schools by mail, took approximately 35 minutes to complete. Teachers were instructed to administer the questionnaires in a standardized fashion to students present at day of assessment.

Smokeless tobacco and cigarette daily use rates were determined separately by respondents' selection of one of 10 use-rate categories. Separate questions assessed "chewing leaf tobacco" and "dipping oral snuff." Previous focus group discussions had indicated that few adolescents used leaf tobacco, but nevertheless called both forms "chew." Although data were collected concerning brand names purchased and used, the general terms "smokeless tobacco" or "chew" best capture student terminology in this sample.

Due to school-required anonymity and the extensive sample size desired, no biochemical verification procedures were employed and no attempt was made to track absentees or dropouts, making these data comparable to earlier surveys. Accordingly, younger students may have underreported tobacco use more than older students and smokeless tobacco may have been underreported more than cigarettes. We received 7,457 usable questionnaires from 106 schools from 59 towns.

Results

Cigarette Use

As expected, there was a positive linear grade trend for smoking peaking at grade 11. Averaged across all grades, boys (11.2 percent) and girls (13.8 percent) reported smoking cigarettes daily at statistically different rates (see Table 1). The reversal from the boys' higher daily use rates in grades 7–9 (9.1 percent versus girls' 7.6 percent) to grades 10–12 (boys, 13.2 percent; girls, 20.7 percent) reflects a significant sex by grade interaction.

Smokeless Tobacco Use

Only 5.7 percent of girls had ever tried smokeless tobacco compared to 36.7 percent of the boys, a reliably large difference. Daily use by girls across the entire sample was only 0.3 percent, while daily use for boys across all grades averaged 5.5 percent, reaching 8.0 percent by grade 12. Weekly rates, although higher, paralleled daily rates. For boys, the higher the grade, the higher the daily smokeless tobacco use.

Total Tobacco Use

Total daily tobacco use, defined as daily use of either cigarettes or smokeless tobacco, increased significantly by grade, peaking at grade 11. Averaged across all grades, total tobacco use was comparable for girls (14.1 percent) and boys (14.8 percent). Like cigarette use, total tobacco use was

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*Rothrock JK: Smokeless Tobacco. Unpublished manuscript, January 1985.

TABLE 1—Percent Daily Substance Use by Adolescents as a Function of Substance, Sex, and School Grade

	Grades						
Sex	7	8	9	10	11	12	Total
Male							
Cigarettes Only	6.2	6.4	11.0	10.8	13.6	11.1	9.4
Smokeless Only	1.3	3.6	4.4	3.0	4.3	6.8	3.6
Cigarette and Smokeless	.7	1.7	2.7	2.5	2.8	1.2	1.8
Total Tobacco	8.2	11.7	18.1	16.3	20.7	19.1	14.8
Total (*n*)	(839)	(698)	(528)	(591)	(508)	(503)	(3,667)
Female							
Cigarettes Only	4.1	8.1	10.8	18.0	23.0	21.1	13.8
Smokeless Only	.4	.3	.5	.0	.3	.2	.3
Cigarette and Smokeless	.0	.1	.0	.0	.0	.0	.0
Total Tobacco	4.5	8.5	11.3	18.0	23.3	21.3	14.1
Total (*n*)	(702)	(670)	(627)	(606)	(640)	(545)	(3,790)
Total							
Cigarettes Only	5.3	7.2	10.9	14.5	18.8	16.3	11.6
Smokeless Only	.9	2.0	2.3	1.5	2.1	3.3	1.9
Cigarette and Smokeless	.4	1.0	1.2	1.3	1.2	.6	.9
Total Tobacco	6.6	10.2	14.4	17.3	22.1	20.2	14.4
Total (*n*)	(1,541)	(1,368)	(1,155)	(1,197)	(1,148)	(1,048)	(7,457)

NOTE: Use of a cigarette is derived in the text by combining the rate of cigarette use with cigarette plus smokeless tobacco use. Smokeless tobacco use is calculated in analogous fashion.

higher for boys (12.0 percent) than girls (8.0 percent) for grades 7–9, while girls' use (20.9 percent) was higher than boys (18.5 percent) for grades 10–12.

Discussion

Boys' cigarette use seemed to fluctuate less by grade than did girls' use. However, between one-fourth and one-half of total daily tobacco use by boys is from smokeless tobacco. As noted in previous research,[14] the lower overall use of tobacco in grade 12 might be accounted for by a higher school dropout rate for tobacco users. Data from Connecticut, a relatively low-use state for smokeless tobacco, suggest previous underestimation of the extent of smokeless tobacco use among adolescent boys in the Northeast. This implies that the national reduction and comparatively low rate of boys' cigarette smoking over the past decade may have been compensated for by an increase in smokeless tobacco use. In fact, boys' total tobacco use may have been comparable to girls' use over these years.

Accordingly, school-based educational efforts need to focus on use of both substances. Questionnaire responses indicated that smokeless tobacco is popular because of the belief that "it is less harmful than cigarettes," "does not hurt others," and "can be done anywhere." While health education may have had an impact on knowledge and attitudes concerning cigarette smoking, smokeless tobacco use is thus not defined by male adolescents as equally dangerous. In Connecticut, legislation to restrict school tobacco use has rarely included smokeless tobacco.

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1T

Mentholated Cigarette Use among Multiphasic Examinees, 1979–86

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Abstract: Mentholated cigarette use was studied in relation to age and race in 29,037 current smokers who were Kaiser Permanente Medical Care Program members. The percentages of mentholated cigarette users were much higher in Blacks and Asians than in Whites, especially in the younger age groups. A marked inverse relationship between mentholated cigarette use and age was present in Blacks and Asians; mentholated cigarette use showed little difference with age in Whites. (*Am J Public Health* 1989; 79:1415–1416.)

Introduction

For a prospective cohort study to determine the health effects of smoking low-yield cigarettes, information was collected from 1979 to 1986 about the smoking habits of members of Kaiser Permanente Medical Care Program who participated in multiphasic health check-ups. Data from this study regarding mentholated cigarette use were analyzed for this report to determine whether previous reports of greater mentholated cigarette use in Blacks would be confirmed in our study population and to study age trends.[1,2] Data regarding Asians are included in this report because of the substantial number of Asian smokers in the study population.

Methods

An automated multiphasic health checkup has been offered since 1964 to about 25,000 persons per year at the Kaiser Permanente Medical Care Program medical centers in Oakland and San Francisco.[3] Starting in July 1979, examinees were asked to complete a supplementary questionnaire that explored their smoking habits in detail.[4] In mid-1980 the check-up was discontinued at the San Francisco facility. By the end of 1986, the questionnaire had been completed by 114,934 examinees (approximately 86 percent of the examinees), of whom 31,428 (27.3 percent) were current smokers. Information on whether or not the cigarettes were mentholated was available for 92 percent of the current smokers. The tar yield was obtained from Federal Trade Commission estimates for each brand and type of cigarette.

If subjects had more than one check-up, only the first was included in this analysis. Age-adjusted proportions of mentholated cigarette users were obtained by the direct method, with the age distribution of the entire study population used as the standard. Mentholated cigarette use habits were examined in the 29,037 current smokers ages 15–79 years of Black, White, or Asian race.

Results

The sex, race, and age distribution of the current smokers is shown in Table 1. The percentage of mentholated cigarette use was higher in women than in men in all age groups for Blacks and Whites, and in most age groups for Asians (Figure 1). There was a marked inverse relationship between age and mentholated cigarette use in Blacks and in Asians, while there was relatively little difference in mentholated cigarette use with age in Whites. Blacks had the highest age-adjusted proportion of mentholated cigarette users (55.5 percent for females, 49.9 percent for males), Asians the second highest (47.9 percent, females, 35.7 percent, males), and Whites the lowest (27.1 percent, females, 18.1 percent, males).

The mean number of cigarettes smoked per day (adjusted for age, race, and sex) was slightly lower in mentholated than in nonmentholated users: 16.2 versus 17.1; 95% confidence interval of the difference = −1.2, −0.7. Mentholated cigarette users were similar to nonmentholated users in their reports of how often they inhaled, depth of inhalation, and how much of the cigarette they smoked. Alhough the mean tar yield of mentholated cigarettes was lower than that of nonmentholated cigarettes for both Blacks (15.5 mg vs 16.0 mg) and Whites (13.8 mg vs 14.0 mg), the mean tar yield of cigarettes smoked by Blacks (adjusted for age and sex) was greater than that of Whites (15.9 mg vs 14.1 mg, 95% CI of the difference 1.7, 2.0).

The switching patterns between mentholated and nonmentholated brands were examined in the 1,688 Black smokers under the age of 40 years for whom follow-up information regarding smoking habits was obtained by a mailed questionnaire (mean interval between baseline questionnaire and follow-up was 4.5 years). Follow-up information was obtained on a slightly greater proportion of nonmentholated than mentholated cigarette users (32 vs 28 percent). The percentage of quitters was slightly higher in nonmentholated users than in mentholated users (17.5 vs 15.4 percent), while the percentage of switchers (from nonmentholated to mentholated or vice-versa) was much higher in the nonmentholated users (14.6 vs 3.6 percent). Using a proportional hazards model to adjust for age and sex and to account for varying length of follow-up, nonmentholated users were 4.2 times more likely to switch than mentholated users (95% CI 2.8, 6.4), while quitting was equally likely in both groups.

Discussion

As others have reported, we found a much higher proportion of mentholated cigarette use in Black smokers than in White smokers. There is a very strong inverse relationship between age and mentholated cigarette use in Blacks. The data suggest that

TABLE 1—Number of Current Cigarette Smokers by Age, Sex, and Race

	Age (years)							
	15–19	20–29	30–39	40–49	50–59	60–69	70–79	Total
Male								
Black	86	777	1323	1031	678	342	57	4294
White	108	1275	2346	1442	1219	769	193	7352
Asian	23	272	454	286	187	74	14	1310
Female								
Black	249	1816	2046	1238	776	302	32	6459
White	238	1914	2441	1523	1452	1023	252	8843
Asian	35	242	216	140	103	42	1	779

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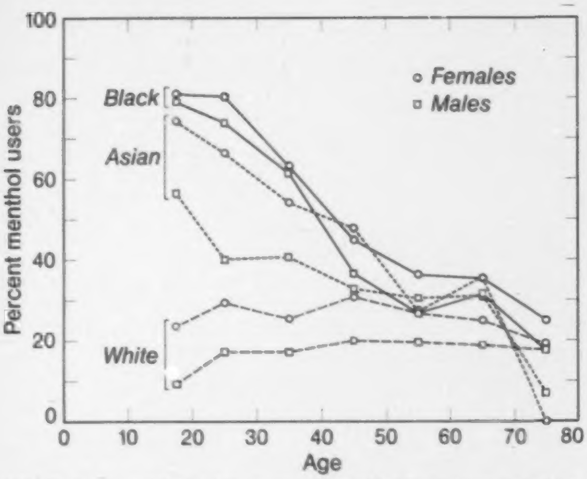

FIGURE 1—Percent Menthol Users among Current Cigarette Smokers by Race, Sex, and Age

the steep decline in the proportion of mentholated cigarette users with age in Blacks can be explained predominantly as a cohort effect, since the findings regarding switching from cigarettes of one mentholation status to another in this study suggest that the prevalence of mentholated cigarette use increases over time in younger Black smokers.

There are few data on the health effects of mentholated cigarettes. A recent case-control of esophageal cancer showed no significant change in risk due to mentholated cigarette use.[5] Our findings have potentially important implications for research into the health effects of mentholated cigarettes. The rates of some smoking-related cancers (e.g., esophagus, lung) are more common in Blacks, at least in part because Blacks are more likely to be smokers.[4] A theoretical basis for a causal relationship between mentholation and cancer exists in that menthol combustion produces carcinogenic compounds such as benzo[a]pyrenes.[5] It has also been hypothesized that the cooling sensation due to menthol may allow deeper, prolonged inhalation of cigarette smoke, thereby increasing the effective yield of tar and other toxic chemicals from the cigarette.[5] This study did not show self-reported differences in inhalation habits between Blacks and Whites, but that may well be due to the anesthetizing effects of menthol.

If mentholation does cause cancer, it may contribute substantially to the excess incidence of certain smoking-related cancers in Blacks relative to Whites. A potential consequence of the very large differences in the prevalence of mentholated cigarette use in younger Blacks relative to Whites would be a widening of the difference between Blacks and Whites of these incidence rates in the future, when younger Blacks reach the higher risk age range for cancer.

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2T

State-Specific Progress Toward the 1990 Objective for the Nation for Cigarette Smoking Prevalence

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Abstract: We predicted the smoking prevalences for 1990 for each state in the US, assuming that the decline in each state from 1985–1990 would be the same as the decline in the US from 1965–1985. In 1985, only three states had smoking prevalences less than 25 percent. Based upon the observed decline in smoking in the US from 1965–1985 of 0.5 percent per year, we predict that only seven states will have smoking prevalences less than 25 percent by 1990. States need to consider current smoking prevalence and achievable rates of decline when setting objectives for 1990 and beyond. (*Am J Public Health* 1989; 79:1416–1419.)

Introduction

As part of its 1990 Health Objectives for the Nation, the US Public Health Service has established a goal that by 1990 the proportion of adults who smoke should be reduced to less than 25 percent.[1] A recently published midcourse review indicated that this objective likely will be met.[2] Despite the apparent success at the national level, however, the variation in the prevalence of smoking from state to state[3] suggests that a prevalence of smoking of less than 25 percent may not be an achievable objective for all states. To address this issue, we predicted 1990 smoking prevalences for all 50 states and the District of Columbia by applying a range of estimates of decline in smoking prevalence to the observed state-specific smoking prevalences for 1985.

Methods

The state-specific smoking prevalence estimates were based on data obtained in September 1985 from the Current

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Population Survey conducted by the Bureau of the Census.[4] In the Current Population Survey, 60,000 households were surveyed in 1,973 counties, cities, and minor civil divisions in the United States. The data were weighted by age, sex, and race to represent the total civilian, non-institutionalized population of each state. To have estimates comparable with previously published national data, we included only persons 20 years of age or older. Current smokers were defined as persons who had smoked 100 or more cigarettes in their lifetimes and who currently smoked. When respondents were not available at the time of the household interview (59 percent of men, 27 percent of women, 42 percent overall), their smoking status was provided by proxy.

We used data from the National Health Interview Surveys from 1965, 1976, 1980, and 1985 to estimate various rates of decline in smoking prevalence among all adults 20 years of age and older. These estimates were age-adjusted to the 1985 US adult population.[5] A current smoker was defined as a person who had smoked at least 100 cigarettes and who smoked at the time of the survey. Proxy responses were not used in the National Health Interview Surveys.

The annual rate of decline in smoking prevalence from 1965–1985 was applied to each state's smoking prevalence in 1985, to project a smoking prevalence for 1990. To account for possible variability in the rate of decline from state to state, we performed a sensitivity analysis. We similarly projected state-specific smoking prevalences for 1990 using 2 times and ½ times the observed rate of decline from 1965–1985. This range in the rate of decline was subsequently compared to the rates of decline in each inter-survey period (1965–76, 1965–80, 1976–80, 1976–85, and 1980–85) in the US. We used the actual state prevalence estimates in all calculations, rounded to the nearest percent for presentation.

Results

In the United States between 1965 and 1985, the age-adjusted prevalence of smoking among adults 20 years of age or older decreased from 40.4 to 30.4 percent, a rate of decline of 0.50 percent per year. In 1985, state-specific smoking prevalences ranged from 15 percent in Utah to 37 percent in Nevada; only three states had smoking prevalences less than 25 percent (Figure 1). If the observed annual rate of decline in the United States from 1965–1985 occurs in each state from 1985–1990, only four additional states will have smoking prevalences less than 25 percent by 1990.

The results of the sensitivity analysis are presented in Table 1. If the decline from 1985–1990 is one-half the observed decline from 1965–1985 (0.25 percent per year), then only three states will have prevalences less than than 25 percent by 1990. Conversely, if the annual decline from 1985–1990 is two times the observed national decline from 1965–1985 (1.0 percent per year), then 25 states will have prevalences less than 25 percent.

The lower bound of the estimate for decline in smoking used in the sensitivity analysis (0.25 percent per year) is less than any decline observed between 1965–1985 (0.39 percent per year between 1965–1976). Similarly, the upper bound of the estimate (1 percent per year) is greater than any decline observed during that period (0.7 percent per year from 1976–1980). When these are applied to the 1985 US smoking prevalence, we predict a smoking rate for the US in 1990 of 27.9 percent (range 25.4 percent to 29.1 percent) (Figure 2).

Discussion

Using national data on the decline in smoking prevalence, we predicted the prevalence of smoking for each state for 1990. We have shown that few states will have smoking prevalences less than 25 percent by 1990, assuming that the decline observed in the United States from 1965–1985 occurs

TABLE 1—State-Specific Smoking Prevalences in 1985 and Predicted for 1990

		Annual Decline in Smoking from 1985–1990[*]		
		.25%/year	.5%/year	1%/year
	Smoking prevalence in 1985[**]	Projected Smoking Prevalence in 1990		
States	(%)	(%)	(%)	(%)
Alabama	30	29	28	25
Alaska	36	35	34	31
Arizona	30	28	27	25
Arkansas	31	29	28	26
California	26	25	23[†]	21[†]
Colorado	29	28	26	24[†]
Connecticut	29	28	27	24[†]
Delaware	32	31	29	27
District of Columbia	31	30	29	26
Florida	31	29	28	26
Georgia	32	31	30	27
Hawaii	27	26	25	22[†]
Idaho	24[†]	23[†]	21[†]	19[†]
Illinois	29	28	26	24[†]
Indiana	33	32	31	28
Iowa	28	26	25	23[†]
Kansas	30	29	27	25
Kentucky	36	35	33	31
Louisiana	29	28	27	24[†]
Maine	29	28	27	24[†]
Maryland	30	29	27	25
Massachusetts	28	27	26	23[†]
Michigan	34	33	32	29
Minnesota	29	28	26	24[†]
Mississippi	31	30	28	26
Missouri	28	26	25	23[†]
Montana	26	25	23[†]	21[†]
Nebraska	24[†]	23[†]	22[†]	19[†]
Nevada	37	36	34	32
New Hampshire	31	29	28	26
New Jersey	28	26	25	23[†]
New Mexico	28	27	26	23[†]
New York	29	27	26	24[†]
North Carolina	32	30	29	27
North Dakota	27	25	24[†]	22[†]
Ohio	32	31	30	27
Oklahoma	33	32	30	28
Oregon	27	26	25	22[†]
Pennsylvania	29	27	26	24[†]
Rhode Island	34	33	32	29
South Carolina	27	26	25	22[†]
South Dakota	28	27	26	23[†]
Tennessee	31	29	28	26
Texas	31	30	28	26
Utah	15[†]	14[†]	12[†]	10[†]
Vermont	31	29	28	26
Virginia	33	32	31	28
Washington	28	27	26	23[†]
West Virginia	33	32	31	28
Wisconsin	26	25	24[†]	21[†]
Wyoming	32	31	30	27
States below 25% (number)	(3)	(3)	(7)	(25)

[*] Trend data from the National Health Interview Surveys were used to estimate the range in rates of decline in smoking for each state from 1985 to 1990. The 1990 projected prevalences were made using the actual 1985 state-specific prevalence but are rounded to the nearest percent for presentation.

[**] The prevalences for 1985 are from the 1985 Current Population Surveys, for adults 20 years of age or older.

[†] Smoking prevalence less than 25 percent.

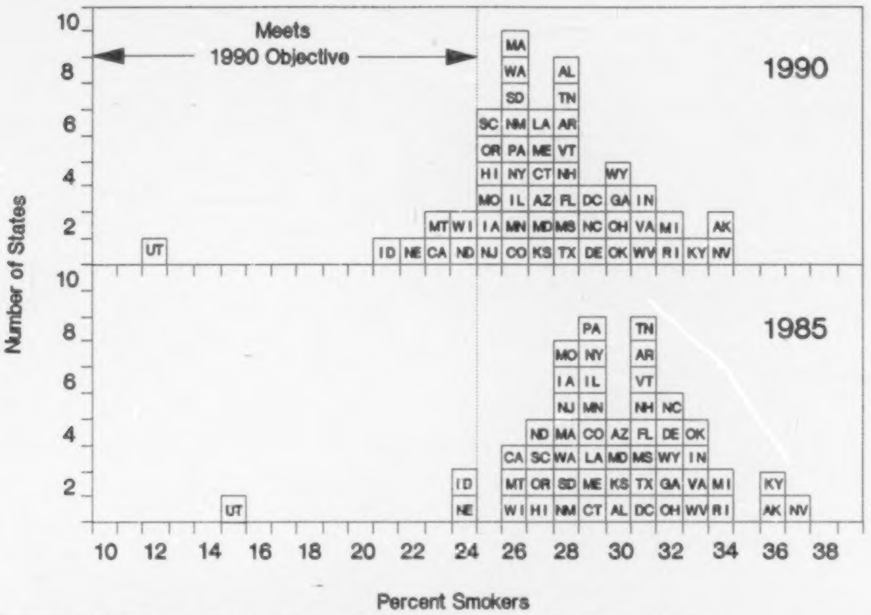

FIGURE 1—Distribution of State-Specific Smoking Prevalences in 1985 and Projected for 1990
Data for 1985 are from the Current Population Surveys for adults 20 years of age or greater. Rates for 1990 are projected assuming a 0.5% annual decline in smoking in each state from 1985–1990. Each square is identified with its 2-digit state abbreviation.

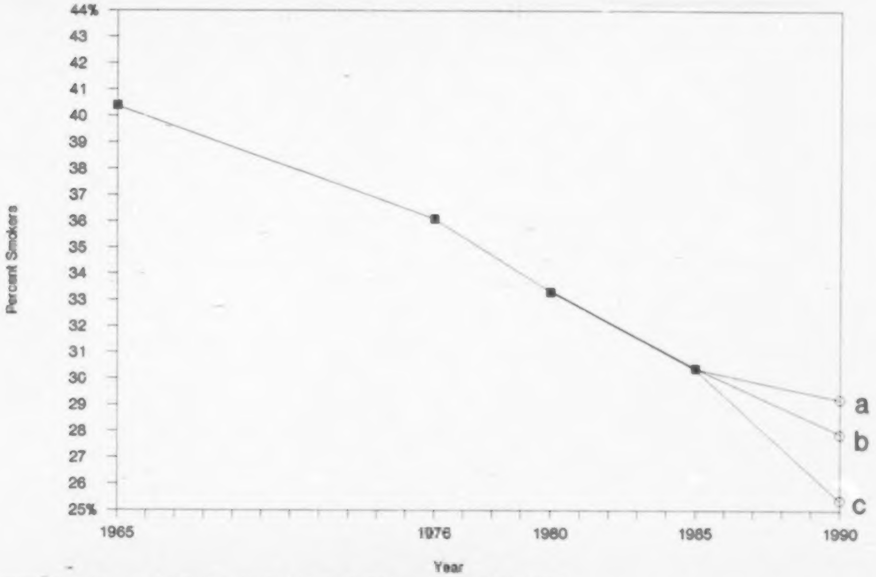

FIGURE 2—Trends in Smoking in the US, 1965–1985 and Projected to 1990
Data for 1965, 1976, 1980, and 1985 are from the National Health Interview Surveys for adults 20 years of age or greater, and are age-adjusted to the 1985 U.S. population. Rates for 1990 are projected assuming an annual rate of decline of 0.25%/year (a), 0.5%/year (b), and 1.0%/year (c), respectively.

in each state from 1985–1990. In fact, in contrast to the conclusion of the Midcourse Review, our analysis suggests that it is unlikely the prevalence of smoking will be less than 25 percent in the United States overall by 1990.

When setting objectives for cigarette smoking prevalence for 1990 and beyond, it is important to consider the methods that will be used to monitor progress toward achieving that objective. For example, although the surveys used in this report were both in-person surveys, slightly different methods were used to assess the prevalence of smoking. In the

TABLE 2—Trends in the Prevalence of Smoking for Adults 20 years of age or greater, United States, 1965–1985

Year	Percent Smokers	Percent Decline/Year from 1965	from 1976	from 1980
1965	40.4	—	—	—
1976	36.1	.39	—	—
1980	33.3	.47	.70	—
1985	30.4	.50	.63	.58

Data are from the National Health Interview Surveys for adults 20 years of age or greater, and are age-adjusted to the 1985 US Census[5]

National Health Interview Survey, respondents were directly questioned about their smoking habits, whereas in the Current Population Surveys, proxy responses were commonly used to determine their smoking status (59 percent of the responses among men were provided by proxy).

When possible, it is important to use state-specific information on trends in smoking when setting objectives. Many states now use data from the Behavioral Risk Factor Surveillance System to estimate the prevalence of smoking.[6] However, telephone surveys generally provide smoking estimates lower than those obtained from in-person surveys. The prevalence estimates from the 22 states that conducted random digit-dialed (self-respondent only) telephone surveys as part of the Behavioral Risk Factor Surveillance System in 1985 were, on the average, 2 percent to 3 percent less than the in-person estimates provided in this report.[3] These lower estimates likely result from telephone noncoverage and lower response rates among smokers in telephone surveys.[7] Nevertheless, this surveillance system has proven useful to states and should be used in the future to set objectives and monitor trends in smoking if these biases are accounted for.

Although predicting smoking prevalences in any state five years hence is difficult, this paper provides a range of estimates that states may consider when setting their own objectives. Because many programs to reduce the prevalence of cigarette smoking are implemented at the state level, it is important for each state to set reasonable objectives for the future.[8] One of the purposes in setting forth an objective is to promote new initiatives. Therefore, selecting a target that will be reached without intervention is inappropriate. Similarly, setting objectives that are clearly not achievable is equally inappropriate. To this end, we hope that this report encourages setting objectives based on a careful review of available data and an assessment of the likely impact of future intervention efforts.

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The News on Smoking: Newspaper Coverage of Smoking and Health in Australia, 1987–88

SIMON CHAPMAN, PHD

Abstract: All newspaper articles (n = 1601) mentioning tobacco or smoking drawn from a 12-month census of 30 Australian capital city newspapers in 1987–88 were coded for content, "slant," and apparent origin. Overall, 62 percent of articles were positive in their orientation toward smoking control objectives, 17 percent were negative, and 21 percent were neutral. Newspapers owned by Rupert Murdoch published 55.7 percent positive and 23.7 percent negative articles compared to 68.0 percent positive and 13.7 percent negative articles in the other large chain of Australian newspapers. (*Am J Public Health* 1989; 79:1419–1421.)

Introduction

Male smoking prevalence has declined in Australia from an estimated 72 percent in 1945[1] to a 1986 level of 31.9 percent.[2] Female smoking prevalence has risen slightly in the same period from 26 percent[1] to 28.8 percent.[2] Almost all of the commentary on this decline in male smoking rates has been preoccupied with the impact of government-sponsored and health agency-sponsored mass health information and persuasion campaigns,[3] and the introduction of smoking control legislation and regulation.[4–7]

Since 1983, most States in Australia have conducted mass-media-based public awareness campaigns although the largest of these efforts has had a budget considerably less

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than the typical cigarette advertising campaign.

Preoccupation with the impact of defined public sector interventions—such as purchased media time and space, school programs, and smoking cessation efforts—has tended to obscure the impact of a principal means by which the public is exposed to messages about smoking: the news media. This study reports a content analysis of a census of all coverage of smoking issues in capital city newspapers in Australia in a sample period in 1987–88.

Methods

All articles dealing exclusively with or mentioning tobacco or smoking published in all 30 Australian capital city newspapers were obtained from a commercial press clipping service for the 12 months May 1987–April 1988. The newspapers included "quality" and popularist newspapers and Sunday editions. A total of 1,601 clippings were thus obtained.

Coding

Each clipping was coded for the publication in which it appeared; date of publication; type of item (news item, feature article, news brief, letter, editorial); apparent origin; principal content; and its "slant" or orientation. These classifications are described in the Appendix. To assess whether the coder's ratings for slant were idiosyncratic, a randomly selected subsample of 1 percent of the clippings (16 articles), was coded by nine peope working in four different smoking control agencies around Australia; a Cohen's Kappa of 0.84 was obtained, indicating high inter-coder reliability (external validity).[8]

Table 1 shows the principal content of all articles, cross-tabulated by slant. News and commentary on the December 1987 ban on smoking on all domestic airlines (187 items), and on the introduction of the Victorian Tobacco Control Act (106 items) which banned all outdoor and cinema tobacco advertising in that State and raised State tax on tobacco, were widely covered. Articles on passive smoking appeared at 125 percent greater frequency than the next most common principal category (advertising and promotion).

When articles were classified by type of article and slant (Table 2), there was a similar distribution for all main categories with the predictable exception of the fewer neutral letters.

Newspaper ownership in Australia is dominated by two main groups, News Corporation owned by Mr Rupert Murdoch, and John Fairfax and Sons. Positive articles in Murdoch-owned papers (55.7 percent) were less common, compared to Fairfax-owned (68 percent) and other-owned (62.2 percent); negative articles were more common in Murdoch papers (23.7 percent), compared to 13.7 percent in Fairfax-owned and 17.2 percent in other-owned.*

TABLE 1—Principal Content of Tobacco-Related Articles by Slant

Slant of Article	Total	Positive (%)	Negative (%)	Neutral (%)
Passive smoking	534	319 (59.4)	117 (21.4)	98 (18.2)
Advertising/promotion	237	136 (53.3)	46 (18.0)	55 (21.6)
Cessation	114	97 (85.1)	5 (4.4)	12 (10.5)
Health issues	215	175 (81.4)	19 (8.8)	19 (8.8)
Taxation	212	88 (41.5)	54 (25.5)	71 (33.5)
Miscellaneous	289	182 (63.4)	34 (11.2)	73 (25.4)

Highest ranking issues: Smoking on aircraft (187), Victorian legislation commentary (106), workplace smoking bans (78), sporting sponsorship by tobacco (75), overviews on smoking and health (66), smoking on public transport (63), calls to ban tobacco advertising (58), how to stop smoking (54). Full breakdown available from author.

TABLE 2—Slant of Tobacco Articles by Type of Publication

Type	Positive (%)	Negative (%)	Neutral or N/A (%)
Article	280 (62.9)	51 (11.5)	114 (25.6)
Feature	70 (61.4)	12 (10.5)	32 (28.1)
News brief	252 (59.9)	56 (13.3)	114 (26.8)
Letter	302 (64.4)	137 (29.2)	30 (6.4)
Editorial	6 (50.0)	4 (33.3)	2 (16.7)
Contributor Column	1 (20.0)	2 (40.0)	2 (40.0)
Cartoon	13 (38.2)	9 (26.4)	12 (35.3)
Advice column	8 (100)	—	—
Part of wider article	65 (69.9)	5 (5.4)	22 (24.7)
Totals	997 (62.3)	276 (17.2)	328 (20.5)

$\chi^2 = 119.91$ ($p < 0.001$ 2df)

When articles were classified by apparent origin and slant (Table 3), the expected polarizations toward positive articles generated or written by health workers, agencies, and physicians, and negative articles inspired or written by the tobacco and advertising industries were found.

Discussion

Because this study examined only one year of publications in one country, no longitudinal or international comparisons can be made. Comparisons of prevalence with articles on drugs or alcohol would require explicit but debatable hypotheses about some ideal level of equivalence or difference and tone of reporting about various topics. In the absence of any reasonable or accepted standards of what ought to be the frequency of tobacco reporting, little can be concluded, other than to suggest that any notion that Australian newspapers are loathe to print items that may offend their tobacco advertisers carries little support.

Evidence from the USA,[9–12] and Australia[13] has demonstrated that there is an inverse relation between the acceptance of tobacco advertising by magazines and the appearance of articles on tobacco. Because all of the newspapers in this study accepted tobacco advertisements, this is not the case with this sample of newspapers in Australia. While Australian newspapers in the past have leaned more toward a negative coverage of proposed bans on tobacco advertising,[14] the evidence here shows no support for the "gag of the tobacco dollar" view. Despite the Murdoch-owned newspapers printing more negative articles than those

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*A full breakdown for all 30 newspapers is available from the author.

TABLE 3—Origin of Article by Slant of Article

Origin	Number	Positive (%)	Negative (%)	Neutral or N/A (%)
No obvious source	553	267 (48.3)	70 (12.7)	196 (35.4)
Doctor/health worker/agency	291	258 (88.7)	13 (4.5)	20 (6.9)
Tobacco or advertising industry	65	—	52 (80.0)	13 (20.0)
Citizen	404	255 (63.1)	119 (29.5)	30 (7.4)
Politician	187	131 (70.1)	8 (4.3)	48 (25.7)
Wire service	121	86 (71.1)	14 (11.6)	21 (17.4)
Total	1601	997 (62.3)	276 (17.2)	328 (20.5)

owned by other publishers, Murdoch newspapers still published 2.4 times as many positive articles as negative.

Explanations of the difference between the apparent editorial or owners' policies with US magazines and Australian newspapers require further study, but may lie in two directions: First, articles in magazines are often planned well in advance and therefore may be open to considerably more explicit or latent censorship than those submitted by journalists in the more immediate world of newspaper writing. However, this view would appear to hold little credence in this study: 114 feature-length articles on tobacco were coded, of which 61 percent were positive and only 11 percent negative, the lowest negative proportion for any major category of article. Like magazine journalism, feature article writing in newspapers is often directed from editorial level down to journalists, so the evidence suggests a newspaper editorial climate conducive to the production of "good news" on smoking in Australia.

The second explanation is that tobacco advertising is more economically important to magazines than to newspapers and that magazines would be thus less inclined to "bite the hand that feeds them." *The Age* newspaper, which ran many more articles on tobacco than any other in the sample, receives about $1.00 from cigarette advertising for every $200 from all advertising,[15] compared to about $1 in $60 for newspapers generally.[16] Australian magazines, by contrast, receive about twice the total proportion of advertising dollars from tobacco as do newspapers.[16] This explanation is more consistent with the greater and more positive coverage of smoking issues in newspapers compared to that in magazines.

APPENDIX A

Coding Classification

Principal content was first coded liberally, and then subsumed into six principal classifications (passive smoking, advertising and promotion, smoking cessation, health issues, taxation, and miscellaneous). "Origin" refers to the apparent origin of the item. News items appear after a process of selection by journalists and editors about what is newsworthy. This selection occurs from sources which frequently include groups with explicit interests in having certain views of issues appear in print. Both the promotion and control of smoking are issues which have vested interest groups keen on having the media publicize news and commentary favorable to their respective interests.[17] A 1973 study of nearly 3,000 items published in the *New York Times* and the *Washington Post* found that 58.2 percent of stories came from "routine" channels (press releases, semi-official documents etc).[18] The concept of "pronunciamento"[17]—or the direct transfer of information, ideas or accusations from interest groups to readers via virtually unaltered transcripts of their press releases—appeared to be applicable to many items in the sample.

"Slant," coded either positive, negative, or neutral/not applicable, refers to the extent to which any article could reasonably be said to be supportive of a social climate antithetical to smoking. Such judgment begs the question of who is doing the judging.[19] I approached this by considering how a person concerned to advance smoking control objectives would judge any given article. Would a smoking control advocate be pleased or displeased to see a particular article appearing in print? Articles judged as being likely to be read by a person favorably disposed toward smoking control, as enhancing the general view that smoking and those institutions, interests, laws and policies that support it are undesirable were coded "positive." Articles that would be understood by such a person as conveying a view of smoking as in some way positive, relatively unimportant compared to other issues, or which were critical of laws, policies and individuals advocating restricting or opposing smoking were coded "negative". Articles which were judged as being neither clearly positive or negative, which evidenced a clear attempt at balancing health and tobacco industry views, or which were simple announcements about (for example) a forthcoming rise in tobacco excise, were coded "neutral/not applicable."

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Influenza Immunization in the Elderly: Knowledge and Attitudes Do Not Explain Physician Behavior

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Abstract: The records of 847 elderly clinic patients were reviewed and the 95 primary care physicians who managed their care were surveyed to determine factors related to their ordering of influenza immunization. Overall, 41 percent of the eligible patients were offered vaccine (range 0–90 percent). Physician offering of vaccine was unrelated to patient age, sex, or race but was higher in patients seen on multiple visits (47 vs 39 percent) and in those vaccinated during the prior year (61 vs 35 percent). Physician offering of vaccine was not associated with perceived incidence of severe side effects or estimates of vaccine efficacy. (*Am J Public Health* 1989; 79:1422–1424.)

Introduction

The elderly have been shown to be at high risk of death and serious illness following influenza infection.[1,2] The Centers for Disease Control[3] and the American College of Physicians[4] have recommended that routine annual influenza immunization be offered to all patients age 65 and older. Such immunization has been shown to be a cost-effective preventive health practice.[5–7] However, less than 20 percent of those over age 65 or otherwise considered to be at high risk receive influenza vaccination each year.[8]

Previous studies have evaluated factors influencing patients' decisions to receive or refuse influenza vaccine,[9–12] but there is little information about what affects physicians decisions to offer vaccination. Physicians appear to know the indications for use of the vaccination, but fail to translate this knowledge into clinical practice.[13]

We studied the frequency and determinants of influenza immunization to see whether differences in physician attitudes, knowledge, or perceived side effects of the vaccine could account for the variation in immunization practices.

Methods

All patients age 65 and older seen in the Primary Care Clinic at the Milwaukee County Medical Complex from October through December of 1984 were identified and their medical records reviewed to determine if they were offered influenza vaccination. Patients were considered eligible if they had not already been vaccinated that season, had no history of egg anaphylaxis or severe reaction to prior vaccination, and did not have an acute febrile illness. Immunization was considered to have been offered by the physician if an order was written or if the patient's refusal of immunization was noted. Demographic data, history of influenza vaccination the prior year, and the type of patient visit (continuing care or visit to a different physician) were also recorded.

Following the study period, a telephone survey was conducted by a non-medical interviewer of all 95 physicians (90 residents and five faculty) who provided direct patient care. Rates of offering influenza vaccine were calculated for the 82 physicians (77 residents and five faculty) who saw at least five eligible patients during the study period. Categorical data were analyzed using the chi square statistic and correlation coefficients (Pearson) were calculated for continuous variables.

Results

The charts of 847 (99.2 percent) of the 854 patients age 65 years and older seen during the study period were reviewed; 812 were eligible for influenza immunization. Overall, 332 (41 percent) of those eligible were offered vaccine; however, individual physicians varied widely in their rates of offering vaccine to their patients, with a range of 0–90 percent. Vaccination was refused by 9 percent of those to whom it was offered. Patient demographics, clinic visit characteristics, and their relation to being offered vaccine are shown in Table 1. Vaccination during the prior calendar year was the strongest determinant of being offered vaccine.

The telephone-administered questionnaire was completed by 88/90 residents and four of five faculty (97 percent overall). Over 80 percent knew the recommended time of year to vaccinate, that influenza vaccine can be given simultaneously with pneumococcal vaccine, and that the objective of influenza immunization is to reduce morbidity and mortality. More than 75 percent correctly responded that influenza immunization is contraindicated in the presence of an acute febrile illness or history of egg anaphylaxis. However, 40 percent incorrectly thought that use of systemic steroids contraindicated vaccination.

Many physicians overestimated the occurrence of rare or unrecognized side effects. Anaphylaxis was estimated to occur >1 percent of the time by 25 percent of physicians, and Guillain-Barré syndrome was believed to occur in >1 percent of recipients by 15 percent of physicians. No association was found between vaccine offering rates and knowledge of incidence rates for anaphylaxis or Guillain-Barré syndrome.* Most physicians (69 percent) considered influenza vaccine to be 70–90 percent effective in preventing infection, but estimates of high vaccine efficacy were not correlated with increased offering rates.

Table 2 shows physician attitudes towards immunizing individuals with various high-risk conditions. The category "age 65 or over" received fewer highest strength recommendations than categories of chronic obstructive pulmonary disease, asthma, residency in a nursing home, renal insufficiency and diabetes. The strength of conviction to immunize

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*Data available on request to author.

TABLE 1—Patient and Clinic Visit Characteristics: Relation to Vaccine Use

Category	N	N (%) Offered Vacine	Odds Ratio (95% CI)	Adjusted Odds Ratio* (95% CI)
Sex				
Female	569	242 (43)	1.26	1.14
Male	243	90 (37)	(.91, 1.73)	(.97, 1.34)
Race				
Black	416	181 (44)	1.25†	1.09
White	376	145 (39)	(.93, 1.67)	(.94, 1.33)
Other	20	6 (30)		
Age (years)				
65–74	465	196 (42)	1.13	1.06
≥75	347	136 (39)	(.84, 1.52)	(.93, 1.22)
Number of visits				
1	601	233 (39)	.72	.85
2 or more	211	99 (47)	(.52, .995)	(.72, 1.01)
Provider seen				
Usual	639	272 (43)	1.4	1.12
New	173	60 (35)	(.97, 2.0)	(.93, 1.35)
Vaccinated in prior year				
Yes	191	117 (61)	2.99	1.73
No/unknown	621	215 (35)	(2.1, 4.2)	(1.49, 2.04)

*Adjusted by multivariate logistic regression
†Black vs all others

TABLE 2—Percentage Distribution of Strength of Physicians' Recommendations to Vaccinate by Patient Risk Factors

Risk Factor	Percent of Physicians Indicating Category of Recommendation*				
	5	4	3	2	1
Chronic Obstructive Pulmonary Disease	84	10	5	1	1
Asthma	77	11	9	2	1
Nursing Home Resident	73	15	8	1	3
Renal Insufficiency	59	16	17	7	1
Diabetes	55	24	16	1	3
Age >65 years	36	26	34	3	1
Heart Failure	29	15	35	10	11
Anemia	8	9	54	12	17

*5 = very strongly, 1 = not recommended

elderly patients was only weakly correlated with offering rates (r = .297, p = .004).

Discussion

Physicians may not use influenza vaccine because of uncertainty about its indications, doubts about its efficacy, or failure to consider vaccination during patient encounters.[14] The recent report by Setia, *et al*,[13] based on 14 physicians, found that only 33 percent of 540 nursing home patients were immunized despite their physicians' strong beliefs in the efficacy and low risk of vaccination. Our study confirms that physicians' perceptions of vaccine efficacy and their strength of conviction to offer vaccination were high but still were not strongly associated with higher rates of vaccination.

While not correlated with the use of vaccine, the perceived incidence of severe vaccine side effects was markedly higher than the actual incidence reported in the literature. Anaphylaxis is an extremely rare occurrence after influenza vaccination,[15] and Guillain-Barré syndrome has not been associated with vaccination since the swine flu vaccine of 1976.[16–18] Educational efforts should emphasize the low risk of major vaccine side effects.

The overwhelming majority of physicians understood key recommendations concerning vaccine use and accurately assessed its efficacy. Other investigators[19,20] have noted that knowledge of the efficacy and recommended use of influenza vaccine does not guarantee its appropriate administration. Moreover, Cohen[20] found that educational seminars for physicians were less important than patient chart-based checklist reminders in changing vaccination behavior.

We recognize several limitations of our findings. This study was conducted in an academic hospital-based outpatient clinic and the results may not be generalizable to the community setting. Certain patients could have declined receipt of the vaccine following discussions with their physicians or have informed the physician that they received the vaccine elsewhere without documentation in the chart. However, given the relatively high offering rate and the fact that such failures would underrepresent the intent to immunize, it is unlikely that this occurred frequently. As this clinic is the sole source of care for most of this indigent population, it is unlikely that many received the vaccine from alternate sites in the community.

Attempts to increase vaccine use should focus on measures other than changing physician attitudes or increasing their knowledge about the vaccine. Strategies which do not rely on individual physician decision-making should be developed to increase appropriate vaccination of the elderly.

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RWJ Foundation Grants Target Children with Mental Illness

Eleven states and the District of Columbia were selected this summer to launch a national initiative to improve services for US children with serious mental illness. The grants were awarded by the Robert Wood Johnson Foundation under its $20.4 million Mental Health Services Program for Youth.

Selected to receive one-year planning grants of $100,000 under the initial phase of the program are: California, the District of Columbia, Kentucky, Michigan, North Carolina, North Dakota, Ohio, Oregon, Pennsylvania, Vermont, Washington, and Wisconsin. Based on the strength of their plans to better coordinate and finance home- and community-based services for mentally ill children, up to eight of the projects will be awarded four-year grants of up to $2.4 million to carry out their plans. The initiative is intended to improve care for the estimated three million US children whose mental disorders—ranging from autism to depression—typically result in long-term disability or institutionalization, foundation officials said.

Directed for the foundation by Mary Jane England, MD, vice president of medical services at the Prudential Insurance Company in Roseland, NJ, the five-year program will allow states to focus their efforts on communities or geographic areas ranging in population from 300,000 to 600,000. The grants require collaboration among the many state and local agencies responsible for planning, providing and financing mental health care for children.

Since the Foundation was established as a national philanthropy in 1972, it has awarded more than $996 million in grants to improve the health of adolescents, children, the elderly, the homeless, the mentally ill, people with AIDS and others. For further information, contact the: Robert Wood Johnson Foundation, College Road, PO Box 2316, Princeton, NJ 08543-2316. Tel: (609) 452-8701.

1T

Occupational Exposure to Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) among Embalmers: A Pilot Seroprevalence Study

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Abstract: We performed a serosurvey of 133 embalmers in an urban area where human immunodeficiency virus (HIV) infection is prevalent. Although we found histories of needlesticks to be common, and the seropositivity rate of hepatitis B virus (HBV) (13%) was approximately twice that of a blood donor comparison group, HIV antibody was uniformly absent in 129 embalmers who denied HIV risk factors, and present in one of four with self-described risk behaviors. The risk of HBV infection was higher among embalmers who have worked more than 10 years, relative risk (RR) 16.2 (95% confidence interval 2.1, 126.5), did not routinely wear gloves, RR 9.8 (CI 3.4, 28.5), or are employed in the city of Boston, RR 4.7 (CI 1.8, 12.0). (*Am J Public Health* 1989; 79:1425–1426.)

Introduction

There is widespread concern in the health care community about occupational exposure to human immunodeficiency virus (HIV). The occupational groups at risk of exposure are those who are also at risk of hepatitis B virus (HBV) infection. Although a substantial number of health care workers have been examined for the risk of both infections, limited information is available on the risk of HBV infection[1,2] and none is available on the risk of HIV infection to embalmers.

Previous studies have shown that HBV has greater potential for nosocomial spread than HIV.[3–7] The purpose of this pilot study is to evaluate the risk of transmission of both infections in relation to work practices in a group of embalmers working in eastern Massachusetts.

Methods

The target population was composed of embalmers from the greater Boston and Worcester, Massachusetts, metropolitan areas. Embalmers were eligible for enrollment if, in their career, they had embalmed 20 or more bodies. The data were collected over a seven-month period, from August 1986 to February 1987.

An oral questionnaire elicited demographic information regarding age, sex, ethnicity, marital status, past medical history, occupational history, and the risk factors for HBV and HIV infection: history of renal dialysis, hepatitis, cancer, bleeding disorder, blood transfusion, receipt of globulin preparation or hepatitis B vaccine, tattoos, and intravenous drug use.

All serum samples were tested for hepatitis B surface antigen (HBsAg), hepatitis B surface antibody (HBsAb), and hepatitis B core antibody (HBcAb) by enzyme-linked immunosorbent assay (ELISA) (Abbott Laboratories, North Chicago, Illinois). Antibody to HIV was measured with the Abbott HTLV-III/LAV ELISA (Abbott Laboratories, North Chicago, Illinois). A repeatedly reactive sample was confirmed by the Western blot technique. Sera that tested positive for HBcAb were screened for antibody to the delta antigen (Abbott Anti-Delta).

Comparison of the seroprevalence among subgroups of the population are expressed as relative risks (RR), estimated by the prevalence odds ratios, with their 95 percent confidence intervals.[8] Variables shown to be risk factors for HBV were included in a logistic regression procedure (Statistical Analysis System Inc., Cary, NC) to evaluate potential confounding.

Results

Of 108 study subjects who had no history of hepatitis B vaccination, 14 (13 percent) had serologic evidence of HBV infection. Of these, HBsAg (with HBcAb) was detected in one, HBcAb alone in one, and HBsAb in 12. Of the 12 with HBsAb in serum, 11 had HBcAb as well. Therefore, the frequency of HBcAb in all participants, including those with any marker, was 9.7 percent (13 of 133). None of the study subjects with serologic markers for HBV infection had evidence of coinfection with the delta virus.

Twenty-five embalmers reported being vaccinated with hepatitis B vaccine. None of the vaccinees had evidence of infection with HBV (HBsAg or HBcAb) and will therefore be considered "seronegative" for purposes of the following analysis.

Years of employment, age, and number of bodies embalmed were significantly associated with HBV infection (Table 1). When these interrelated variables were entered into a logistic regression procedure, employment for more than 10 years correlated most strongly with prior infection (adjusted odds ratio = 85.9); no additional predictive value was added to the regression model by age or number of bodies embalmed.

Embalmers who do not routinely wear gloves were almost 10 times more likely to have serologic markers of HBV infection than those who do. Controlling for duration of employment strengthened this association (adjusted odds ratio = 16.7). No correlation was found between HBV markers and reported use of gowns or aprons, shoe coverings, goggles, glasses, or face masks.

Subjects employed in Boston were more likely to have evidence of HBV infection than those employed outside of Boston. After adjusting for years of employment and use of gloves, Boston's embalmers were 12 times more likely to have HBV markers than others. All subjects with evidence of previous infection with HBV reported sustaining one or more accidental needlesticks during their career. However, the

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TABLE 1—Risk Factors for HBV Infection

Variables	Total	Serologic Evidence of HBV Infection	Relative Risk (95% CI)
Employed > 10 years as embalmer	66	13 (20)	16.2 (2.1, 126.5)
Embalmed ≥ 1000 bodies	67	12 (18)	6.9 (1.5, 32.8)
Do not always wear gloves	18	7 (39)	9.8 (3.4, 28.5)
Employment in Boston	37	9 (24)	4.7 (1.8, 12.0)
Greater than 35 years of age	67	11 (16)	3.6 (1.2, 11.2)
Eat or drink while embalming	48	7 (15)	1.8 (0.66, 4.7)
Smoke while embalming	43	6 (14)	1.6 (0.50, 4.3)

*Figures in parentheses denote percentages.

number of known HBV cases embalmed or recognized HBV needlesticks was not related to HBV infection. Three of the four subjects who disclosed non-occupational risk factors had evidence of previous HBV infection.

When information on history of previous employment in other occupations involving blood exposure was analyzed, there was no observed association with prevalence of HBV markers. Twenty-one percent of embalmers with HBV markers reported having clinically overt hepatitis in the past.

All 133 subjects were tested for antibodies to HIV. One of four self-described members of recognized high-risk groups tested positive for HIVAb; all other subjects were seronegative.

Discussion

During the embalming procedure, embalmers often come into contact with large amounts of blood as the vascular system is drained. Depending on the cause of death and whether an autopsy has been performed, they may be required to handle various body parts and tissues, to make numerous incisions and subsequently suture the incised tissue. However, compared to other health care professions, embalmers have been largely ignored by researchers evaluating the risk of occupationally transmitted infections. The results of this study indicate that embalmers are at increased risk of infection with HBV when compared to the general population,[9] but have not shown increased risk of HIV infection. Overall, the seroprevalence of hepatitis B among health care workers ranges from 5 to 32 percent.[9–15] The 13 percent prevalence of hepatitis B markers among the non-vaccinated subjects indicates that embalmers are a moderately high-risk group of health care professionals. The finding that 79 percent of these individuals had simultaneous HBcAb and HBsAb is consistent with the results of Dienstag and Ryan,[9] who reported that frequent blood exposure, and in general more intense exposure, was correlated with the prevalence of both antibodies.

Recently concern over exposure to asymptomatic HIV carriers has motivated improved infection control practices among health care workers. Embalmers currently maintain these standards for known AIDS (acquired immunodeficiency syndrome) cases, but need additional education to emphasize adherence to universal precautions. The Centers for Disease Control recommends the routine use of gloves, masks, protective eyewear, gowns, and waterproof aprons for all persons performing or assisting in postmortem procedures.[16]

The study participants recalled having embalmed a total of 298 AIDS cases, compared to 285 confirmed AIDS deaths in Massachusetts when the study was initiated. This comparison suggests that the estimates of occupational exposure to AIDS cases are accurate.

Little emphasis has been placed by this occupational group on the risk of exposure to HBV in the workplace. Because this study demonstrates that the risk of occupationally acquired HIV infection is extremely low, and the risk of occupationally acquired HBV infection is substantial, emphasis should be placed on prevention of transmission of HBV during embalming by the use of universal blood and body fluid precautions,[16] and routine immunization with hepatitis B vaccine.

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